PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA

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DECLARATION BY THE CANDIDATE

I hereby declare that the thesis entitled **"PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA"** submitted towards partial fulfillment of the requirements for the award of **DM CARDIOLOGY** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Shibba Takkar Chhabra,** Professor, Department of Cardiology, **Dr. Ashima Taneja,** Professor & Head, Department of Obst. & Gynae, & **Dr. Suman Puri**, Professor, Department of Obst. & Gynae. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

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my family, friends

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LIST OF ABBREVIATIONS

PPCM- peripartum cardiomyopathy

RHD- rheumatic heart disease

CHD- congenital heart disease

LSCS- lower segment caesarean section

PIH- pregnancy induced hypertension

NYHA- new york heart association

EF- ejection fraction

LVEDD- left ventricular end diastolic dimension

LVESD- left ventricular end systolic dimension

PPH- post-partum hemorrhage

CVD- cardiovascular disease

IHD-ischemic heart disease

BMI- body mass index

SVR- systemic vascular resistance

FGR- fetal growth restriction

HDP- hypertensive disorders of pregnancy

MTP- medical termination of pregnancy

ASD- atrial septal defect

VSD- ventricular septal defect

PAH- pulmonary arterial hypertension

DCMP- dilated cardiomyopathy

IUD- intra uterine demise

GTCS- generalised tonic clonic seizures

DM- diabetes mellitus

PASP- pulmonary artery systolic pressures

AKI- acute kidney injury

ACE- angiotensin converting enzyme

ARNI- angiotensin receptor neprilysin inhibitor

MRA- mineralocorticoid receptor antagonist

ABSTRACT

Introduction: Cardiovascular disease complicates 1–4% of pregnancies. Pregnancy is marked by the rapid onset of many hemodynamic and physiological changes to accommodate the needs of the growing fetus. Pregnancy can exacerbate an already existing cardiovascular illness leading to suboptimal maternal and foetal outcomes.

Aims and objectives: We aimed to study the prevalence, clinical profile, risk factors & materno-fetal outcomes in pregnant females with cardiovascular ailments.

Materials and methods: Single centre, prospective observational study was conducted on 135 subjects and their short term follow up was done for 6 months post-partum.

Results: Mean age and gestational period of participants was 32.24 years and 33.14 weeks respectively. Among those, PPCM was the commonest (38.5%) followed by RHD (11.8%) and CHD (6.66%). LSCS was common mode of delivery in most patients (74.8%). Preterm birth was more common in PPCM patients with PIH than without (p value 0.009). Significant recovery predictors in PPCM patients were NYHA class, BNP levels, baseline EF, LVEDD, and mean gestational period. Maternal mortality occurred in 8.14% of our individuals and was substantially correlated with severe baseline symptoms, post-partum diagnosis, the presence of sepsis and postpartum hemorrhage (PPH), and low hemoglobin levels.

Conclusion: Low and middle-income nations should prioritize a cardio-obstetrics team approach in healthcare. Timely diagnosis and pre-conception counseling, providing lesion specific counseling and care, and formulating tailored risk scores and guidelines.

Keywords: Pregnancy, Materno-fetal outcomes, Sepsis, Mortality.

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KEY TO MASTER CHART

Y-	YES
N-	NO
PPCM-	PERIPARTUM CARDIOMYOPATHY
HDP-	HYPERTENSIVE DISORDERS OF PREGNANCY
RHD-	RHEUMATIC HEART DISEASE
CHD-	CONGENITAL HEART DISEASE
VTE-	VENOUS THROMBOEMBOLISM
G-	GRAVIDA
P-	PARAGRAVIDA
L-	LIVE BIRTH
A-	ABORTION
NSR-	NORMAL SINUS RHYTHM
GHK-	GLOBAL HYPOKINESIA
H/O-	HISTORY OF
VKA-	VITAMIN K ANTAGONIST

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INTRODUCTION

BACKGROUND:

Based on the contemporary data, CVD affects around 1-4% of pregnancies and accounts for nearly 10-15% of total maternal mortality[1], varying across regions depending on the variable burden of risk factors like congenital heart disease (CHD), ischemic heart disease (IHD), and rheumatic heart disease (RHD).

Medical literature widely acknowledges that pregnancy acts as a stress test for a woman's cardiovascular system. During pregnancy, the cardiovascular system undergoes significant physiological changes that are necessary to support the growing foetus but can unmask or worsen underlying heart conditions. Thus, pregnancy is a critical time for identifying women at risk for future cardiovascular issues.

Although most cardiac conditions are well tolerated during pregnancy with favorable outcomes, some cardiac conditions have significant maternal and fetal morbidity and mortality. Women who experience complications like preeclampsia, gestational hypertension, or peripartum cardiomyopathy are at significantly higher risk of developing cardiovascular diseases later in life.

Many previous studies have described pregnancy as a **"natural stress test"** because it can reveal latent cardiovascular conditions that may otherwise go undetected until later in life. Therefore, Pregnancy is often called a maternal stress test.[2]

Geographical variation: The prevalence and paradigm of antenatal heart disease contrast between high-income, industrialized, and low- and middle-income countries.[3] In developed countries, improved survival of women with congenital heart defects has increased the prevalence of CHD-related complications during pregnancy. Congenital heart disease (CHD) is the most common in Western countries, while Rheumatic heart disease (RHD) is the most common in low- and middle-income countries, accounting for up to 88% to 90% of cases.[4] However, the prevalence of RHD has decreased as healthcare

coverage has increased, and the ratio of RHD to CHD has decreased from 10:1 to 3:1 in developing countries.[5]

Risk Factors for Cardiovascular Ailments in Pregnancy

- Pre-existing Heart Conditions: Women with pre-existing conditions, particularly congenital or acquired heart diseases, are at higher risk.
- Advanced Maternal Age: Women over the age of 35 are at increased risk for developing pregnancy-related cardiovascular complications, including hypertensive disorders and ischemic heart disease.
- Obesity: A higher body mass index (BMI) is associated with an increased risk of hypertension, gestational diabetes, and ischemic heart disease, which all compound cardiovascular risks during pregnancy.
- Hypertension and Preeclampsia: Hypertensive disorders during pregnancy, especially preeclampsia, increase the risk of heart failure, stroke, and long-term cardiovascular disease.
- Diabetes Mellitus: Pre-existing diabetes or gestational diabetes contributes to cardiovascular strain by accelerating atherosclerosis and increasing the risk of ischemic heart disease.
- Smoking and Substance Abuse: Smoking and the use of recreational drugs, such as cocaine, increase the risk of acute myocardial infarction and other cardiovascular events.
- Family History of Cardiovascular Disease: A strong family history of heart disease can predict increased cardiovascular risk during pregnancy.
- Socioeconomic Factors: Women from low-resource settings often have limited access to antenatal care and face a higher burden of cardiovascular disease, particularly RHD.

SPECTRUM OF CARDIAC DISORDERS IN PREGNANCY:

The causes of pregnancy-related cardiovascular diseases are varied and depend on the individual pathology involved. This highlights

prevalent cardiovascular disorders during pregnancy and their proposed aetiologies:

• Cardiomyopathy: Multiple hypotheses exist concerning the origin of this disease process. Hemodynamic instability, autoimmune etiology, viral myocarditis, and microchimerism are the most common explanations.[6] The common risk factors for cardiomyopathy in non-pregnant adults also persist during pregnancy and the postpartum period, including alcohol consumption, doxorubicin administration, and the misuse of substances such as cocaine and methamphetamines.[7]

• Coronary artery disease: Pregnant women share the same etiology as non-pregnant women for ischemic heart disease. These people are at risk for ischemic heart disease due to risk factors such as diabetes mellitus, obesity, smoking, physical inactivity, hypertension, hyperlipidemia, and hypertriglyceridemia.[8]

• Pregnancy associated Myocardial infarction: The risk factors for myocardial infarction connected with pregnancy are the same as those for coronary artery disease. Preeclampsia and eclampsia are two pregnancy conditions that have been linked to myocardial infarction.[9]

• Valvular disease: Although pregnancy's hemodynamics may make some valve problems worse, it's unclear if pregnancy contributes significantly to the development of recently discovered pregnancy-related valvular disorders.

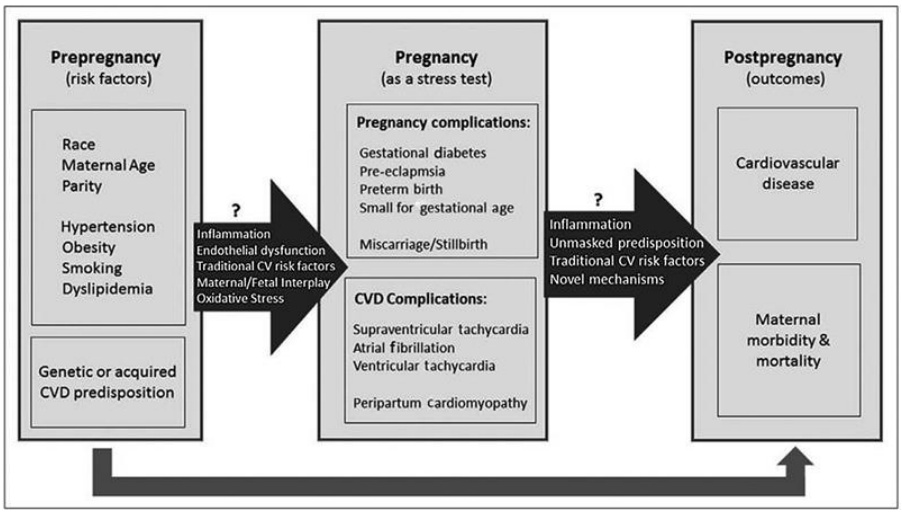

FIGURE 1: PREDISPOSING ETIOLOGIES OF CARDIOVASCULAR DISORDERS AMONG PREGNANT FEMALES.[10]

CARDIOVASCULAR CHANGES DURING PREGNANCY:

The fundamental physiology of pregnancy and the associated alterations contribute to the promotion of cardiac disease processes. Pregnant women commence experiencing these physiological changes as early as 5 weeks. The alterations and adaptations may differ among individuals, with many of these changes stemming from the placenta's attachment to the uterine walls, which triggers the release of hormones and subsequent modifications in maternal physiology.[11-15]

TABLE 1: PHYSIOLOGICAL CHANGES DURING PREGNANCY

Cardiovascular Parameter	Physiological Change	Timing/Peak
Blood Volume	Increases by 30-50% to support fetal growth and prepare for delivery blood loss.	Peaks at 32 weeks gestation.
Cardiac Output	Increases by 30-50% due to both increased heart rate and stroke volume.	Peaks in the second trimester and stays elevated until delivery.
Heart Rate	Increases by 10-20 beats per minute to meet increased circulatory demands.	Gradual increase starting in the first trimester, peaks by third trimester.
Stroke Volume	Increases by 10-30% due to increased blood volume.	Highest during the second trimester.
Systemic Vascular Resistance (SVR)	Decreases by 20-30% due to the vasodilatory effects of progesterone a elaxin.	Begins early in pregnancy, with the lowest point in mid-pregnancy.
Blood Pressure	Decreases slightly in early pregnancy due to reduced SVR, then returns to pre-pregnancy levels by the third trimester.	Lowest during the second trimester; returns to baseline by third trimester.
Venous Return	Decreases, especially when lying flat (supine), due to compression of the inferior vena cava by the growing uterus (supine hypotension).	Most evident in the third trimester.
Heart Position	The heart is displaced slightly upward and to the left due to the expanding uterus, which can affect the electrical axis.	Occurs gradually as the uterus enlarges.
Cardiac Hypertrophy	Mild increase in heart size due to increased workload and volume overload.	Develops over the course of pregnancy.
Oxygen Consumption	Increases by 20-30% to meet the metabolic demands of the mother and fetus.	Begins early and continues to increase throughout pregnancy.

Above discussed physiological and hormonal changes are hypothesized as contributing factors to cause decompensated states of

pregnancy with pre-existing heart disease, such as cardiomyopathy, congenital heart diseases, and valvular disease.

Maternal and Fetal Outcomes in Pregnant Women with Cardiovascular Disease

• Maternal Outcomes:

- ❖ Heart failure: Women with significant cardiovascular problems, such as severe valvular disease or cardiomyopathy, are at an increased risk of heart failure during pregnancy, delivery, or the postpartum period.
- ❖ Arrhythmias and Thromboembolic Events: Pregnancy induces a pro-thrombotic condition, elevating the risk of arrhythmias and thromboembolic incidents, such as stroke and pulmonary embolism, in women with cardiovascular disease.
- ❖ Maternal Mortality: Cardiovascular disease is a predominant cause of maternal fatalities, especially in women with inadequately managed pre-existing illnesses.
- ❖ Birth Complications: The decision between vaginal and cesarean birth is determined by the severity of the cardiac condition. Cardiovascular insufficiency may need premature delivery or cesarean procedure.

• Obstetric and Fetal Outcomes:

- ❖ Preterm Birth: Maternal cardiovascular disease correlates with a heightened risk of preterm delivery resulting from maternal or fetal impairment.
- ❖ Fetal Growth Restriction (FGR): Diminished uteroplacental blood flow resulting from maternal cardiovascular illness may result in IUGR, affecting the infant's long-term health.
- ❖ Low Birth Weight: Infants born to women with cardiac conditions frequently have reduced birth weights as a result of inadequate placental perfusion.
- ❖ Perinatal Mortality: Severe maternal heart illness elevates the risk of perinatal death, especially in instances of congenital heart abnormalities or compromised maternal cardiac function.

- ❖ Congenital Heart Disease: Women with specific types of congenital heart disease have a heightened risk of having offspring with congenital heart problems, attributable to genetic and environmental influences.

Hypertensive disorders of pregnancy:

Hypertensive disorders of pregnancy (HDP) represent prevalent medical problems during gestation and are a major contributor to maternal and neonatal morbidity and mortality. The primary subtypes of hypertensive diseases in pregnancy include chronic hypertension, gestational hypertension, preeclampsia, and eclampsia.

1. Classification of Hypertensive Disorders of Pregnancy

The hypertensive disorders of pregnancy are categorized into four main types:

A. Chronic Hypertension

- It is defined as high blood pressure (≥140/90 mm Hg) that predates pregnancy or is diagnosed before 20 weeks of gestation. It persists beyond the postpartum period. It may result from primary hypertension (essential hypertension) or secondary causes such as kidney disease or endocrine disorders. Women with chronic hypertension are predisposed to developing superimposed preeclampsia and other pregnancy complications.

B. Gestational Hypertension

- It refers to new-onset increased blood pressure (≥140/90 mm Hg) after 20 weeks of gestation in a previously normotensive woman without the presence of proteinuria or other features of preeclampsia. It usually resolves after delivery but can progress to preeclampsia. The earlier in pregnancy it occurs, the higher the risk for complications.

C. Preeclampsia

- Preeclampsia is a multisystem disorder characterized by new-onset hypertension after 20 weeks of gestation (≥140/90 mm Hg) accompanied by proteinuria (≥300 mg in 24 hours) or other systemic signs such as thrombocytopenia, impaired liver

function, renal insufficiency, pulmonary oedema, or neurological symptoms.

- Severe Preeclampsia: Defined by more severe blood pressure elevations (≥160/110 mm Hg) and/or more significant organ damage.

D. Eclampsia

- Eclampsia is defined as occurrence of generalized tonic-clonic seizures in a woman with preeclampsia that cannot be attributed to other causes. It is a severe complication of preeclampsia and can occur antepartum, intrapartum, or postpartum. Eclampsia is life-threatening and requires urgent medical intervention to prevent severe maternal and foetal outcomes.

E. Chronic Hypertension with Superimposed Preeclampsia

- This condition occurs when a woman with pre-existing chronic hypertension develops signs of preeclampsia, such as proteinuria or a sudden worsening of blood pressure control, during pregnancy.

Diagnostic Criteria for Preeclampsia.[2]

Always necessary (Hypertension)

- **SBP ≥ 140 mm Hg or DBP ≥ 90 mm Hg** on 2 occasions at least 4 hours apart after 20 weeks' gestation in a woman with previously normal blood pressure.
- **SBP ≥ 160 mm Hg or DBP ≥ 110 mm Hg** on 1 occasion.

And one of the following:

Proteinuria

- ≥ 300 mg per 24-hour urine collection (or extrapolated from timed collection), OR
- Protein/creatinine ratio ≥ 0.3 mg/dL, OR
- Dipstick reading of 2+ (used only when other methods are not available).

Or any one of the following (in the absence of proteinuria):

- **Thrombocytopenia**
  Platelet count < 100,000/mm^3.

- **Renal insufficiency**
  Serum creatinine concentration > 1.1 mg/dL or a doubling of serum creatinine concentration in the absence of other renal disease.
- **Impaired liver function**
  Elevated blood levels of liver transaminases to twice normal concentration, or
  Severe persistent right upper quadrant or epigastric pain unresponsive to medication and not accounted for by other diagnoses.
- **Pulmonary edema**
  Diagnosed by physical examination or chest x-ray.
- **Neurological signs**
  New-onset headache unresponsive to medication and not accounted for by alternative diagnoses or visual symptoms, or
  Visual disturbances.
- **Fetal growth restriction**
  Estimated fetal weight < 10th percentile.

2. Pathophysiology of Preeclampsia

- Abnormal Placentation: In preeclampsia, abnormal development of the placental blood vessels leads to poor placental perfusion. This placental hypoxia and ischemia trigger the release of factors that damage maternal endothelial cells.
- Endothelial Dysfunction: Endothelial cell damage leads to widespread vasoconstriction, increased vascular permeability, and activation of the coagulation system, contributing to the characteristic signs of hypertension, proteinuria, and organ dysfunction.
- Inflammation and Immune Dysfunction: Inflammation and immune maladaptation also play a role in the development of preeclampsia.

3. Management of Hypertensive Disorders of Pregnancy
    - Close monitoring of blood pressure and maternal symptoms.
    - Antihypertensive medications (e.g., labetalol, nifedipine, or methyldopa) may be prescribed if blood pressure is severely elevated.
    - Planned delivery at 37–39 weeks is often recommended, depending on disease severity.
    - Immediate administration of magnesium sulphate to prevent recurrent seizures in eclampsia patients.
    - Stabilization of the mother and urgent delivery are usually required.

4. Prevention and Risk Reduction

- Low-dose Aspirin: The use of low-dose aspirin (81 mg daily) starting between 12-16 weeks of gestation has been shown to reduce the risk of preeclampsia in women at high risk.
- Calcium Supplementation: In women with low calcium intake, supplementation has been associated with a reduced risk of hypertensive disorders, particularly in populations with calcium deficiency.
- Lifestyle Interventions: Maintaining a healthy weight, regular physical activity, and controlling pre-existing conditions such as hypertension and diabetes can help reduce the risk of hypertensive disorders in pregnancy.

The notion of a collaborative endeavor to provide patient care is not a new concept, particularly when there is a high risk of morbidity and mortality. The cardio-obstetric team is a multidisciplinary team that includes the following team members at a minimum: obstetrician, maternal-foetal medicine (MFM), cardiology, anesthesia, neonatology, and critical care personnel. The team is composed of the patient, physicians, nurses, and administration. The method is reminiscent of the "heart team" paradigm, which is a traditional approach to the management of the majority of at-risk cardiac surgical patients by a team of cardiologists and cardiothoracic surgeons. [16]

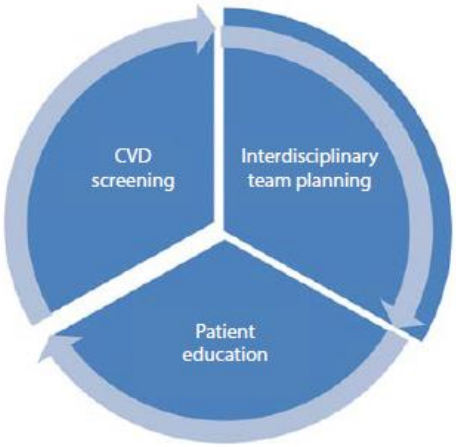

FIGURE 2: MULTIDISCIPLINARY TEAM APPROACH

SCORE PREDICTORS FOR HEART DISEASE IN PREGNANCY:

It is crucial for both the patient and the clinician to be able to predict the incidence and severity of potential complications in order to determine the advisability of avoiding or terminating early pregnancy, the necessity of performing surgery or other therapeutic interventions before pregnancy in the patient with correctable disease, and the establishment of a management plan for pregnancy, labor, and delivery. Siu et al. developed and validated a scoring system (CARPREG [Cardiac Disease in Pregnancy]) approximately two decades ago. This system has been extensively referenced and employed by clinicians to evaluate the risk of pregnancy. The system was founded on information that was readily accessible through a comprehensive history, physical examination, and echocardiogram. This scoring system was especially adequate for risk assessment of patients with congenital, valvular, and myocardial disease, and those with a history of arrhythmias. However, the CARPREG scoring system did not address important risk factors including prosthetic heart valves, aortopathies, and pulmonary arterial hypertension unless they were associated with symptoms or other cardiac events before pregnancy. In this issue of the Journal, Silversides et al. present CARPREG II, a more comprehensive risk scoring system, designed to add high-risk conditions not included in CARPREG I.[17]

Ten predictors were broadly classified into three categories, as per the CARPREG II [18]study:

Five fundamental cardiac factors:

- antecedent cardiac events (history of HF, stroke, or transient ischemic attack) or arrhythmia (three points),
- baseline NYHA functional class III or IV or cyanosis (three points),
- high-risk valve lesions/left ventricular outflow tract obstruction (two points),
- systemic ventricular dysfunction (two points),
- absence of prior cardiac interventions (one point);

Four lesion-specific variables:

- mechanical prosthesis (three points),
- coronary artery disease (two points),
- high-risk aortopathy (two points),
- pulmonary hypertension (two points);

One variable related to the delivery of care: late pregnancy assessment (one point).

Table 2: Modified World Health Organization (WHO) Classification of Maternal Cardiovascular Risk.[19]

WHO Pregnancy Risk Classification (Risk of pregnancy by medical condition)	**Cardiovascular Conditions by WHO Risk Class**

WHO Risk Class I
No detectable increased risk of maternal mortality and no or mild increase in morbidity.
	• Uncomplicated, small or mild
	  ○ Pulmonary stenosis
	  ○ Patient ductus arteriosus
	  ○ Mitral valve prolapse
	• Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage).
	• Atrial or ventricular ectopic beats, isolated

WHO Risk Class II (If otherwise well and uncomplicated) *Small increased risk of maternal mortality or moderate increase in morbidity.*	• Unoperated atrial or ventricular septal defect • Repaired tetralogy of Fallot • Most arrhythmias
WHO Risk Class II or III (Depending on individual) *Risk as indicated in Class II (above) or Class III (below).*	• Mild left ventricular impairment • Hypertrophic cardiomyopathy • Native or tissue valvular heart disease not considered WHO I or IV • Marfan syndrome without aortic dilatation • Aorta <45 mm in aortic disease associated with bicuspid aortic valve • Repaired Coarctation
WHO Risk Class III *Significantly increased risk of maternal mortality or severe morbidity. Expert counseling required. If pregnancy is decided upon, intensive specialist cardiac and obstetric monitoring needed throughout pregnancy, childbirth and the puerperium.*	• Mechanical valve • Systemic right ventricle • Fontan circulation • Cyanotic heart disease (unrepaired) • Other complex congenital heart disease • Aortic dilatation 40-45 mm in Marfan Syndrome • Aortic dilatation 45-50 mm in aortic disease associated with bicuspid aortic valve
WHO Risk Class IV (Pregnancy contraindicated) *Extremely high risk of maternal mortality or severe morbidity; pregnancy contraindicated. If pregnancy occurs termination should be discussed. If pregnancy continues, care as for class III.*	• Pulmonary arterial hypertension of any cause • Severe systemic ventricular dysfunction (LVEF <30%, NYHA III-IV)* • Previous peripartum cardiomyopathy with any residual impairment of left ventricular function • Severe symptomatic mitral or aortic stenosis • Marfan syndrome with aorta dilated >45 mm • Aortic dilation >50 mm in aortic disease associated with bicuspid aortic valve • Native severe Coarctation

AIMS AND OBJECTIVES

1. To study the prevalence of heart diseases in pregnant women in tertiary care centre in north India.
2. To study maternal cardiac, obstetric, and foetal outcomes in pregnant women with heart diseases.
3. To investigate the risk factors associated with morbidity and mortality; and recovery predictors in pregnant women with cardiovascular diseases.

REVIEW OF LITERATURE

A study was conducted at a tertiary care center in Mumbai which predicted adverse maternal outcomes in pregnant women with heart diseases and involved 66 pregnant women with structural heart diseases. Their key findings showed that majority of the women were between 20-35 years of age, and 51.5% were first-time mothers. Vaginal delivery was the most common, but there was a high rate of cesarean sections (41.07%) and instrumental deliveries. The most common cardiac ailment was rheumatic heart disease (59.09%) followed by congenital heart disease (25.75%). The study also revealed that women with CARPREG II scores of four or higher were more likely to experience adverse cardiac events, including maternal mortality and heart failure. Complications like low birth weight and preterm deliveries were frequent. The study determined that the CARPREG II index is proficient in forecasting maternal outcomes and underscores the significance of adept prenatal care in enhancing pregnancy results for women with cardiac conditions. However, it noted that perinatal complications did not strongly correlate with the index, and more studies are needed to validate CARPREG II in the Indian population.[20]

A study examined the short-term cardiovascular impacts of severe pre-eclampsia (PEC), and compared 63 women with PEC to 36 healthy pregnant women using echocardiographic, clinical, and laboratory measurements. Their key findings were that Women with PEC had significantly higher RVSP and reduced right ventricular systolic strain (RVLSS), indicating increased pulmonary pressures. About 13% of PEC patients exhibited diastolic dysfunction, and 9.5% developed pulmonary edema. No significant left-sided systolic dysfunction was observed, but strain parameters indicated subtle abnormalities. The study suggested that these cardiac alterations are early markers of dysfunction in severe pre-eclampsia and may predict acute complications like pulmonary edema.[21]

A study conducted in Jaipur investigated the effects of rheumatic heart disease (RHD) during pregnancy. The study included 90 cases out of 2,378 deliveries, which accounted for 3.78% of the total. Mitral valve stenosis was the most frequently identified condition (46.6%). Congestive cardiac failure was the most prevalent complication, afflicting 33.3% of the participants. Furthermore, the investigation determined that preterm birth was the most prevalent neonatal issue, with 43.3% of cases involving it. The prognosis for women with RHD has improved, despite the challenges they face, highlighting the significance of early detection and management throughout pregnancy.[22]

A study was performed to assess the short-term cardiovascular outcomes of patients with severe pre-eclampsia. It was determined that eighty percent of the cases were late-onset pre-eclampsia, manifesting beyond 34 weeks of gestation, and they may exhibit severe symptoms. The findings indicated that left ventricular mass at 1–3 days postpartum was elevated following severe late-onset pre-eclampsia (57 g/m²) in comparison to normal pregnancy (48 g/m²). The research determined that the cardiac repercussions of severe late-onset pre-eclampsia were minimal and temporary. This indicates that left ventricular hypertrophy after severe late-onset pre-eclampsia may be a secondary physiological reaction to heightened peripheral resistance linked to pre-eclampsia and vascular mechanisms, rather than enduring cardiac hypertrophy postpartum, which may account for the elevated long-term cardiovascular risk following pre-eclampsia.[23]

A retrospective study was conducted at AIIMS, New Delhi which examined fetal and cardiac outcomes in pregnant women with congenital heart disease (CHD) and included a sub-analysis on cyanotic and non-cyanotic defects, as well as operated versus non-operated cases. The study involved a total of 86 pregnant women with CHD, with atrial septal defects being the most common at 29.06%. Among these patients, 31.39% had cyanotic CHD, and approximately

55% had previously undergone surgery for their cardiac defects. In terms of cardiovascular complications, 5.8% of the participants experienced heart failure, 7.0% had pulmonary arterial hypertension, 8.1% were classified as New York Heart Association functional class IV, 9.3% necessitated admission to the intensive care unit, and one case of perinatal mortality was reported. Women with CHD experienced substantially more adverse fetal outcomes than those without heart disease. These included operative vaginal delivery, extended hospital stays, embryonic growth restriction, preterm birth (before 37 weeks), low birth weight (under 2,500 g), a 5-minute APGAR score of less than 7, and admissions to the neonatal intensive care unit. The study concluded that women with CHD face a higher risk of adverse fetal and cardiac outcomes. However, these outcomes can improve with proper pre-conception cardiac optimization, comprehensive antenatal care, and multidisciplinary team management.[24]

A study was conducted to examine maternal deaths due to cardiovascular disease during pregnancy and up to 12 months after delivery in Sri Lanka. Out of 2,855 pregnancy-related deaths; 57.7% confirmed as maternal deaths. Among these, 284 deaths (17.25%) were attributed to heart disease complicating pregnancy. Rheumatic heart disease was identified as the leading cause of heart disease-related maternal deaths, accounting for 21.1% deaths. Other significant contributors included cardiomyopathies (20.7%), and congenital anomalies (12.0%). Additionally, medically contraindicated pregnancies resulted in 54 deaths (19%).[25]

A study was performed in northern India to assess maternal and fetal outcomes in pregnant women with cardiac conditions. Ninety women with cardiac disease, either previously diagnosed or detected during pregnancy, were participated in the study. The incidence of heart disease in all pregnancies at the hospital was determined to be 4.3%. Rheumatic heart disease (RHD) was the predominant cause of cardiac lesions, including 56.6% of patients, whereas congenital heart disease

constituted 13.3%. In women with rheumatic heart disease, mitral stenosis was the predominant lesion, occurring in 23.3% of cases. Among individuals with congenital heart illness, mitral valve prolapse was the most common, accounting for 5 instances. Cardiomyopathy was observed in seven women. Heart failure occurred in 11.1% of cases. A majority of the women (36.7%) underwent cesarean section, 7% experienced assisted instrumental vaginal birth, and 9% had first-trimester abortions. Four maternal fatalities occurred. 85.6% of live births were recorded, and 72.8% of the infants weighed over 2 kg, with no cases of congenital cardiac disease found among the neonates. The study determined that pre-pregnancy diagnosis, counseling, suitable referrals, antenatal monitoring, and delivery at well equipped facilities markedly enhance pregnancy outcomes for moms and infants with heart disease.[26]

A study was done in a tertiary care center of northern India to assess the obstetric and fetal outcomes of women who had valvular heart disease or prosthetic heart valve replacement secondary to RHD. The study included a total of 80 patients, of whom 60 (75%) had native RHD and 20 (25%) underwent mechanical prosthetic valve replacement. A maternal adverse event was observed in 34 cases (42.5%), including 1 mortality (1.2%), 2 new-onset atrial fibrillation (2.5%), 20 balloon mitral valvotomies (25%), 3 mitral valve replacements (3.7%), and 7 heart failure hospitalizations (8.7%). Mitral valve infective endocarditis was observed in one patient (1.2%). Preterm delivery was observed in 19 cases (23.7%), 7 abortions (8.7%), and 1 intrauterine fatality (1.2%). Low birth weight was seen in 53.7%. Consequently, it was determined that women with rheumatic heart disease are at a heightened risk for both the mother and the fetus. [27]

The study by Karen Melchiorre and colleagues investigated the long-term cardiovascular effects of preeclampsia (PE), focusing on left ventricular (LV) dysfunction. Their key Findings were Women who experienced preterm preeclampsia had higher rates of moderate-

severe left ventricular dysfunction and concentric remodeling, compared to those with term preeclampsia and healthy pregnancies. Stage B heart failure (asymptomatic but with structural abnormalities) was observed in 70% of preterm PE cases one year postpartum, compared to only 24% of term PE cases. 40% of women with preterm PE developed essential hypertension within two years, much higher than the 8% incidence in controls. The study concluded that preeclampsia's cardiovascular impact extends beyond pregnancy, with preterm cases being more vulnerable to long-term heart complications. Early cardiovascular risk assessment and monitoring in postpartum women who experienced preeclampsia, especially preterm cases, are crucial for preventing future heart diseases and highlighted the need for targeted interventions to address the heightened cardiovascular risk in these women.[28]

The study by Meriem Koual and colleagues analyzed the short-term effects of preeclampsia on maternal health. The study included 155 patients and focused on their postpartum health outcomes, particularly cardiovascular and renal conditions. Key Findings were Obesity and gestational diabetes were identified as significant risk factors for preeclampsia, affecting 46% and 15% of patients, respectively. Postpartum follow up revealed persistent High blood pressure in 66% of patients immediately after delivery, and 16% still had hypertension three months postpartum, requiring continued antihypertensive treatment. Proteinuria (excessive protein in urine) was found in 66% of patients postpartum, and in 22% at three months, indicating ongoing renal issues. The study highlights that preeclampsia significantly impacts maternal health in the short term, with many patients experiencing persistent hypertension and renal issues postpartum. Continuous monitoring and etiological assessments are essential for managing future pregnancies and mitigating long-term cardiovascular risks.[29]

The aim of this prospective study was to manage patients with a multidisciplinary approach to maternal cardiovascular disease and to evaluate its influence on maternal and fetal outcomes in a tertiary care center in Bihar, India. The study included 65 pregnant women with heart disease, aged between 20 and 35 years, who were compared to a control group of 65 pregnant women hospitalized at the same time without heart disease. The prevalence of heart disease in pregnancy was found to be 5.8%. The most common condition was rheumatic heart disease (RHD), accounting for 62.5%, followed by corrected congenital heart disease (CHD) at 12.5% and non-corrected CHD at 10.9%. Among these patients, 58.5% were classified as NYHA Class I and II, 26.2% as Class III, and 15.4% as Class IV.

The mitral valve was most frequently affected by RHD, involved in 35.3% of cases, followed by the tricuspid valve at 15.0%. Eight patients (1.1%) underwent cardiac surgery for therapeutic reasons. The most common congenital abnormalities observed were ventricular septal defects (VSD) at 3%, followed by atrial septal defects (ASD) at 1.5% and pulmonary ductal atresia (PDA) at 1.5%. Women with heart disease had higher rates of medical termination of pregnancy (MTP), emergency lower segment cesarean sections (LSCS), and instrumental births compared to the controls. In terms of maternal mortality, there were four deaths (6.2%) in the heart disease group, with no maternal deaths in the control group; all deceased women belonged to NYHA Class IV and were anemic. Two women died intrapartum from RHD, while the other two died postpartum due to peripartum cardiomyopathy.

The study found significantly more cases of low-birth-weight infants (36.4%) compared to the control group ($p = 0.001$). Additionally, patients with heart disease experienced a statistically significant higher rate of obstetric complications and had an increased risk of developing anemia, hypertension, hypothyroidism, cholestasis, fetal growth restriction (FGR), and gestational diabetes mellitus (GDM) ($p = 0.017$). Factors such as multiparity, severe valvular lesions, NYHA function class III or IV, arrhythmias, and low ejection fraction were associated with poorer maternal outcomes in this study.[30]

A novel score was generated to predict the left ventricular recovery in patients with peripartum cardiomyopathy. This can be easily applied in clinical practice.[31]

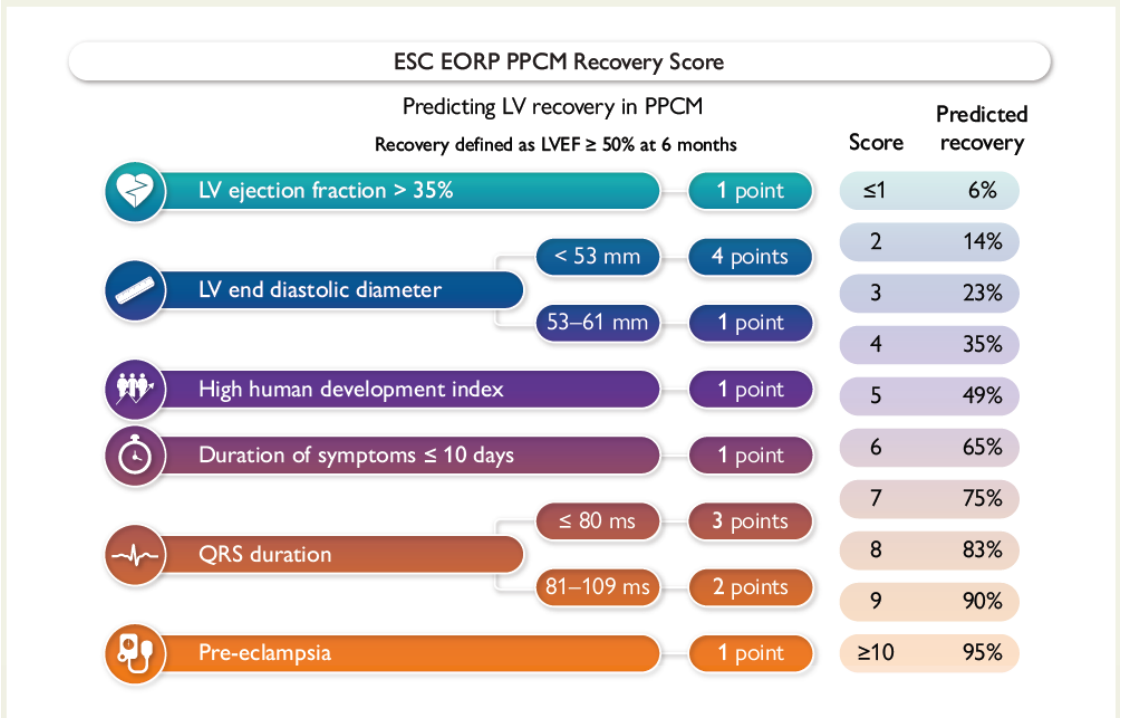

FIGURE 3: ESC EORP PPCM RECOVERY SCORE

A prospective observational study was conducted to determine the short-term outcomes of peripartum cardiomyopathy, focusing on clinical aspects as well as left ventricular systolic function. The study took place in the Department of Cardiology at the Manmohan Cardiothoracic Vascular and Transplant Center, Institute of Medicine, Kathmandu, Nepal, from July 2018 to January 2022. A total of 68 women met the inclusion criteria, with a mean age of 28.38 years (ranging from 19 to 44 years). The most common clinical presentation was dyspnea, followed by lower limb edema. Six patients (8.8%) presented during the last month of pregnancy, while 62 patients (91.2%) presented in the postpartum period. The mean left ventricular ejection fraction at presentation was 30.01%. Full recovery was observed in 60.29% of the patients by the six-month follow-up, and the mortality rate was 4.41%.[32]

A retrospective study was conducted to evaluate the Long-term maternal outcomes of subsequent pregnancies (SSPs) in 47 patients with peripartum cardiomyopathy (PPCM). The clinical and echocardiographic findings were compared between the recovery group (RG) and nonrecovery group (NRG), defined as left ventricular ejection fraction >50% and <50% after an index of pregnancy, respectively. Mean age was 27.0 ± 6.1 years, 80% were of African American descent, and 75.6% from a low socioeconomic background. Thirty (66.7%) women were in the RG. Overall, SSPs were associated with a decrease in mean left ventricular ejection fraction from 45.1% to 41.2%. At 5 years, adverse outcomes were significantly higher in the NRG compared with the RG (53.3% vs 20%;) and Five-year all-cause mortality was 13.33% in the NRG compared with 3.33% in the RG.[33]

In southern India, a prospective cohort study was conducted at a solitary center on patients with peripartum cardiomyopathy (PPCM). The patients were monitored for a period of six months. The primary endpoint was a composite incidence of decompensation-related rehospitalization, all-cause mortality, and poor recovery (defined as a left ventricular ejection fraction (LVEF) of less than 45% at 6 months). The results indicated that the mean LVEF at presentation was 34.7%, and 63.4% of patients experienced complete LV recovery after discharge. The composite endpoint was experienced by 32.5% of the study population, with a high left atrial volume index (LAVi) and a low right ventricular fractional area change (RVFAC) at presentation serving as independent predictors. The use of Inotropic therapy during index hospitalization (with dobutamine or levosimendan) and bromocriptine therapy was not associated with a superior outcome. The study concluded that poor outcomes are predicted by RVFAC (<31.4% with 86% accuracy) and LAVi (>29.6 ml/m2 with 72% accuracy) at presentation, but not by LVEF.[34]

In a study titled “Pregnancy Outcomes in Women with Rheumatic Mitral Valve Disease,” conducted by Iris M. van Hagen et al. in 2018, 390

patients were evaluated. The study found that mitral stenosis (MS), with or without mitral regurgitation (MR), was present in 273 women, while 117 women had isolated MR. The degree of MS was classified as mild in 20.9% of cases, moderate in 39.2%, severe in 19.8%, and the severity was not classified in the remaining cases. One maternal death occurred during pregnancy in a patient with severe MS. Hospital admissions were necessary for 23.1% of the women with MS, primarily due to heart failure: 15.8% of those with mild MS, 23.4% with moderate MS, and 48.1% with severe MS ($P < 0.001$). Heart failure also occurred in 23.1% of patients with moderate or severe MR. During the study, 16 patients required intervention while pregnant: 14 underwent percutaneous balloon mitral commissurotomy, and 2 had surgical valve replacements (one for MS and one for MR). Follow-up at six months postpartum was conducted for 53% of the participants, and three more patients died (one with severe MS, one with moderate MS, and one with moderate to severe MR).[35]

In a meta-analysis by Steinberg et al. (2017), maternal and fetal outcomes in pregnant women with mechanical heart valves (MHVs) were examined across 800 pregnancies from 18 studies. The study assessed maternal outcomes, such as death and thromboembolism, and fetal outcomes, including spontaneous abortion and congenital defects. Various anticoagulation methods were explored:

1. Vitamin K antagonist (VKA) throughout pregnancy
2. Low molecular weight heparin (LMWH) throughout pregnancy
3. LMWH in the first trimester, followed by VKA (LMWH + VKA)
4. Unfractionated heparin in the first trimester, followed by VKA (UFH + VKA)

Maternal risk was lowest with VKA at 5%, compared to 16% for the other methods. However, fetal risk was highest with VKA at 39%, whereas LMWH showed a risk of 13%, with LMWH + VKA at 23% and UFH + VKA at 34%. No significant differences were found in fetal risk between those on ≤5 mg of daily warfarin and those on LMWH.[36]

In a 2017 meta-analysis conducted by Wu et al[,] which examined 258,000 patients with preeclampsia, researchers evaluated the relationship between preeclampsia and the future risk of heart failure, coronary heart disease, composite cardiovascular disease, death due to coronary heart or cardiovascular disease, stroke, and stroke-related death. The study concluded that preeclampsia is associated with a four-fold increase in the future incidence of heart failure and a two-fold elevated risk of coronary heart disease, stroke, and death from coronary or cardiovascular disease, after adjusting for age, body mass index, and diabetes mellitus.[37]

A meta-analysis by S. De Haas et al. in 2017, which reviewed 48 studies from 1997 to 2016, found that during a normotensive pregnancy, most cardiac geometric indices begin to change from the second trimester onward. In these pregnancies, Left Ventricular Mass (LVM) and Relative Wall Thickness (RWT) increase by 20% and 10%, respectively, indicating a concentric rather than eccentric remodeling pattern. However, in hypertensive pregnancies, cardiac adaptation significantly diverges, with a marked 95% increase in LVM and 56% increase in RWT from baseline values, reflecting a more pronounced structural adjustment compared to healthy pregnancies.[38]

In a study conducted by McNamara et al[.] (2015) to evaluate the recovery of left ventricular ejection fraction (LVEF) and clinical outcomes, the multicenter IPAC (Investigations of Pregnancy Associated Cardiomyopathy) study included a total of 100 women with peripartum cardiomyopathy (PPCM). LVEF was assessed through echocardiography at baseline, and at 2, 6, and 12 months postpartum.
The cohort consisted of 30% Black women, 65% White women, and 5% from other racial backgrounds. The mean age of the participants was 30 ± 6 years. Additionally, 88% were receiving beta-blockers, and 81% were on angiotensin-converting enzyme inhibitors or angiotensin receptor blockers. Factors associated with a lower LVEF at 12 months included an initial LVEF of less than 0.30 ($p = 0.001$), left ventricular

end-diastolic dimension (LVEDD) of 6.0 cm or greater ($p < 0.001$), being Black ($p = 0.001$), and presenting after 6 weeks postpartum ($p = 0.02$). Furthermore, survival free from major cardiovascular events—such as death, transplantation, or the need for a left ventricular assist device—was assessed. The study found that 13% of participants experienced major events or persistent severe cardiomyopathy.[39]

Another study named “Pregnancy-related cardiovascular deaths in California: beyond peripartum cardiomyopathy” done by Hameed AB et al[16] concluded that obesity, african american race and substance abuse were important risk factors for pregnancy related cardiovascular deaths. This study retrospectively examined a case series of 64 cardiovascular pregnancy-related deaths from 2002 through 2006 and found that 42 deaths caused by cardiomyopathy, and the pregnancy-related mortality rate from cardiomyopathy was 1.54 per 100,000 births.[40]

The 2007 study by S. S. Brar et al., conducted in California, provided a comprehensive analysis of the incidence, mortality, and racial disparities associated with peripartum cardiomyopathy (PC). This research revealed that the overall incidence of PC was approximately 1 in every 4,025 deliveries, underscoring its rarity in the general population. However, the data highlighted substantial variation across racial groups. African-American women exhibited the highest incidence rate at 1 in 1,421 deliveries, indicating that they were significantly more susceptible to developing PC. Their risk was found to be nearly 2.9 times higher than that of white women, where the incidence was 1 in 4,075 deliveries, and seven times higher than Hispanic women, who had an incidence rate of 1 in 9,861 deliveries. Asian women also had a relatively higher incidence at 1 in 2,675 deliveries compared to white and Hispanic populations, though their risk remained markedly lower than that of African-American women. With a mean follow-up period of 4.7 years, the study employed the Kaplan-Meier method to evaluate survival outcomes. Results showed that 96.7% of patients remained

free from all-cause mortality, suggesting that despite African-American women's increased susceptibility to PC, modern management and therapies might be mitigating the associated mortality. This survival rate contrasts with older studies reporting higher mortality rates, implying that recent advances in heart failure (HF) management—such as beta-blockers, ACE inhibitors, and refined cardiac care protocols—are likely improving outcomes for PC patients. Overall, this population-based study highlights important racial disparities, with African-American women disproportionately affected by PC, possibly due to genetic, socioeconomic, or healthcare access factors that increase vulnerability in this group. The study's findings also indicate that the mortality associated with PC may be lower than previously reported, pointing to the effectiveness of current HF treatment options in improving long-term survival for those diagnosed with PC. This research underscores the need for targeted prevention and early intervention strategies, particularly for African-American women, to mitigate PC risks, as well as the potential benefits of continued advancements in HF therapies and prenatal care to optimize patient outcomes.[41]

In a 2006 study by James AH et al., the incidence, mortality, and risk factors for pregnancy-related acute myocardial infarction (AMI) in the United States were examined. The study identified 859 hospital discharges with a diagnosis of acute myocardial infarction, corresponding to a rate of 6.2 cases per 100,000 deliveries. Among these cases, there were 44 deaths, resulting in a case fatality rate of 5.1%. Although AMI is relatively uncommon in women of reproductive age, pregnancy itself significantly raises the risk—by approximately 3- to 4-fold. The study highlighted several independent risk factors for pregnancy-related AMI, including advanced maternal age (particularly age 30 and above), race, certain medical conditions, and obstetric complications. Key contributing conditions included hypertension, thrombophilia, diabetes mellitus, smoking, transfusion, and postpartum infection. These findings emphasize that while pregnancy-related AMI is rare, the associated risk increases with specific demographic and

clinical factors, underscoring the importance of careful monitoring and preventive strategies for at-risk pregnant women.[42]

A 2022 meta-analysis conducted in South Asia sought to assess the prevalence and variety of heart disease in pregnant women, reflecting the unique challenges and risks in this population. After a thorough screening and selection process, 25 studies were included in the final analysis, offering a comprehensive overview of cardiac health issues affecting pregnant women in the region. The analysis revealed a pooled prevalence of heart disease among pregnant women at 1.46%, highlighting a notable subset of the maternal population at risk. Among these women with heart conditions, rheumatic heart disease was the most common, accounting for 70.25% of cases, while congenital heart disease was present in 18.10%. This high prevalence of rheumatic heart disease emphasizes its continued public health burden in South Asia, despite its decline in many other regions due to improved healthcare interventions. The study also underscored significant adverse pregnancy outcomes associated with maternal heart disease. The pooled prevalence of preterm labor and delivery among pregnant women with heart disease was 17.63%, indicating that heart disease in pregnancy markedly raises the risk of premature birth, which can lead to further complications for both mother and child. Moreover, the meta-analysis reported maternal and fetal mortality rates of 26.14 and 50.48 per 1,000, respectively, among pregnant women with heart disease. These rates reflect a substantial impact on survival, underscoring the critical need for heightened monitoring, early diagnosis, and comprehensive prenatal care tailored to this high-risk group. The findings from this analysis highlight the ongoing public health challenge posed by heart disease during pregnancy in South Asia, with rheumatic heart disease being the most prevalent contributor, suggesting the need for targeted strategies to manage and reduce its impact on maternal and fetal health outcomes in the region.[43]

In a recent retrospective study (2022) conducted in England by Williams et al[,] researchers compared the risk of cardiovascular disease outcomes for women with and without a history of gestational hypertension and pre-eclampsia. The study utilized national hospital admission data and included women who had one or more singleton live births in England between 1997 and 2015. Over 18 years of follow-up, the incidence of total cardiovascular disease (CVD) was noted for three groups: normotensive women, women with prior gestational hypertension, and women with prior pre-eclampsia. The rates were as follows: 57.1 per 100,000 person-years for normotensive women, 85.8 per 100,000 person-years for women with gestational hypertension, and 99.3 per 100,000 person-years for women with pre-eclampsia.

Among specific types of cardiovascular disease, gestational hypertension was strongly linked to dilated cardiomyopathy and unstable angina, while pre-eclampsia was closely associated with hypertrophic cardiomyopathy and acute myocardial infarction.[44]

MATERIALS AND METHODS

SOURCE OF DATA

A Single centre, Prospective observational study was done on pregnant females with heart diseases which were enrolled from department of Cardiology and department of Obstetrics & Gynaecology in Dayanand Medical College and Hospital Ludhiana, Punjab which is a tertiary care hospital in north India.

METHOD OF COLLECTION OF DATA

The data was collected for 18 months starting from January 2023 to June 2024 which included 135 women with heart disease, either previously known or diagnosed during pregnancy. All pregnant women with congenital or acquired cardiac conditions, including arrhythmias, referred to our hospital were included. A comprehensive pre-designed proforma was used to gather relevant information on heart disease in pregnancy. Collected baseline data included age, parity, gestational age, history of prior cardiac events (only post-intervention events were recorded for those who had undergone cardiac procedures), specific cardiac lesions, previous surgeries, cyanosis (oxygen saturation below 90%), and use of cardiac medications. Each patient underwent a thorough clinical examination, including chest and cardiovascular auscultation, a 12-lead ECG, and a transthoracic echocardiogram to assess both left and right ventricular systolic function. The delivery mode—whether vaginal (with or without induction), or LSCS—was carefully documented. Systemic bi-ventricular ejection fraction was calculated using validated methods. Valvular area and gradients and severity of valvular regurgitation was also measured using standardized criteria. All patients were scored according to CARPREG II risk scoring system.

All women underwent cardiac assessment at baseline, and were followed there-after until 6 months postpartum. Pregnant women who underwent termination or had miscarriage (fetal death at <20 weeks gestation) were also included.

OUTCOMES

Cardiac outcomes: The following parameters were studied: cardiac arrest, maternal cardiac death, sustained arrhythmia necessitating treatment, left-sided heart failure (defined as pulmonary edema), right-sided heart failure, stroke or transient ischemic attack, cardiac thromboembolism, myocardial infarction, and vascular dissection.

Obstetric outcomes: Intra-uterine growth retardation, premature membrane rupture (membrane rupture prior to the onset of uterine contractions), premature labor (spontaneous onset of labor prior to 37 weeks of gestation), post-partum hemorrhage (vaginal delivery >500 mL, caesarean delivery >1000 mL or requiring transfusion), and placental abruption and any other related complications were included.

Fetal death (>22 weeks of gestation or >500 g), neonatal death (<30 days post-partum), stillbirths, and congenital heart defects (CHD) in the offspring of a mother with CHD were among the perinatal outcomes.

In addition, these patients were examined for other associations, including pregnancy-induced hypertension [PIH], which is defined as new-onset hypertension (>140/90 on two separate occasions) after more than 20 weeks of gestation, pre-eclampsia (PIH criteria plus >0.3 g proteinuria in the 24-hour urine sample), eclampsia (pre-eclampsia with grand mal seizures), concurrent evidence of sepsis, acute kidney injury, and the need for mechanical ventilation.

Recovery from peripartum cardiomyopathy was defined as recovery of LVEF to ≥50% or increase by more than 20%. As already mentioned, recovery usually occurs between 3 and 6 months postpartum, but might occur as late as 48 months postpartum.[45,46]

STATISTICAL ANALYSIS

Data were described in terms of range; mean ±standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. To determine whether the data were normally distributed, a Kolmogorov-Smirnov test was used. Comparison of quantitative variables between the study groups was done using Mann Whitney *U* test and Kruskal-Wallis Test for independent samples for non-parametric data. For comparing categorical data, Chi square ($\chi 2$) test was performed and fisher exact test was used when the expected frequency is less than 5. A probability value (*p* value) less than 0.05 was considered statistically significant. All statistical calculations were done using (Statistical Package for the Social Science) SPSS 21.0 version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

OBSERVATIONS AND RESULTS

This prospective study was carried out at a tertiary care centre in North India, focusing on all pregnant females with a current or past history of cardiovascular diseases.

During the enrolment period, a total of 1,825 pregnancies resulted in deliveries, with 74.6% by cesarean section and 25.4% by vaginal delivery. Among these, 135 patients experienced some form of cardiovascular ailment. Of the 135 patients with cardiovascular issues, 74.8% were delivered by cesarean section and 25.2% by vaginal delivery.

Of 135 patients; 52 patients were diagnosed as peripartum cardiomyopathy which was the most common cardiovascular ailment in our patients; followed by 45 patients which were diagnosed as Hypertensive Disorders of Pregnancy. 16 and 9 patients had Valvular and congenital heart disease respectively. Out of 16 patients of VHD, 15 had rheumatic aetiology while 1 had mitral valve endocarditis. Thrombotic disorders were observed in total six patients. Among others, 2 patients had idiopathic PAH and 1 had hypertrophic cardiomyopathy and 1 had DCMP (newly diagnosed) and 3 patients with myocarditis.

TABLE 3: COMPARISON OF PATIENT PHENOTYPES ACCORDING TO AGE

All patients were divided into age subgroups as following:

		PPCM		HDP		VHD/CHD/VTE		Total
		No. of cases	%age	No. of cases	%age	No. of cases	%age	
AGE GROUP	< 25	7	13.5%	3	6.7%	2	6.5%	12
	26-30	20	38.5%	10	22.2%	10	32.3%	40
	31-35	16	30.8%	16	35.6%	13	41.9%	45
	36-40	6	11.5%	8	17.8%	4	12.9%	18
	> 40	3	5.8%	8	17.8%	2	6.5%	13
Total		52	100.0%	45	100.0%	31	100.0%	128

PPCM patients were younger as compared to patients with other heart diseases and the most common (38.5%) age group in PPCM patients was 26-30 years while in patients with other heart disease most common age group was 31-35 years.

Mean age of HDP patients was highest (34.33 years) among all the different phenotypes of patients and this was statistically significant finding.

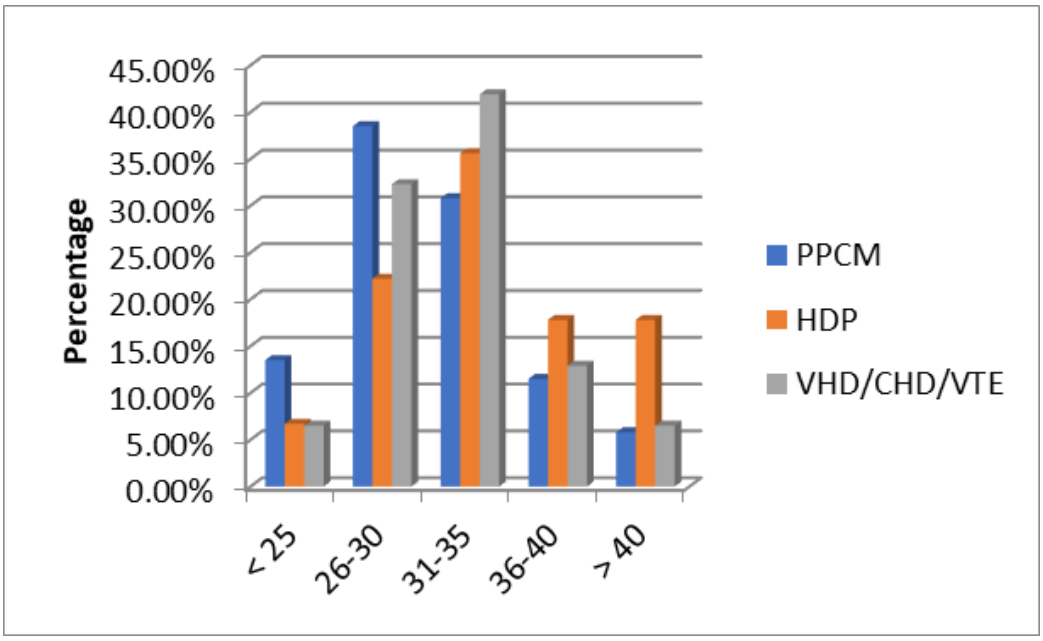

FIGURE 4: COMPARISON OF PATIENT PHENOTYPES ACCORDING TO AGE

TABLE 4: COMPARISON OF PATIENT PHENOTYPES ACCORDING TO BMI

Patients with VHD/CHD/VTE had lower BMI as compared with OOCM and HDP patients; it was found to be a significant association.

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
BMI	< 22.9	1	1.9%	1	2.2%	7	22.6%	9	19.078	0.004
	23-24.9	13	25.0%	7	15.6%	8	25.8%	28		
	25-29.9	33	63.5%	29	64.4%	14	45.2%	76		
	> 30	5	9.6%	8	17.8%	2	6.5%	15		
Total		52	100.0%	45	100.0%	31	100.0%	128		

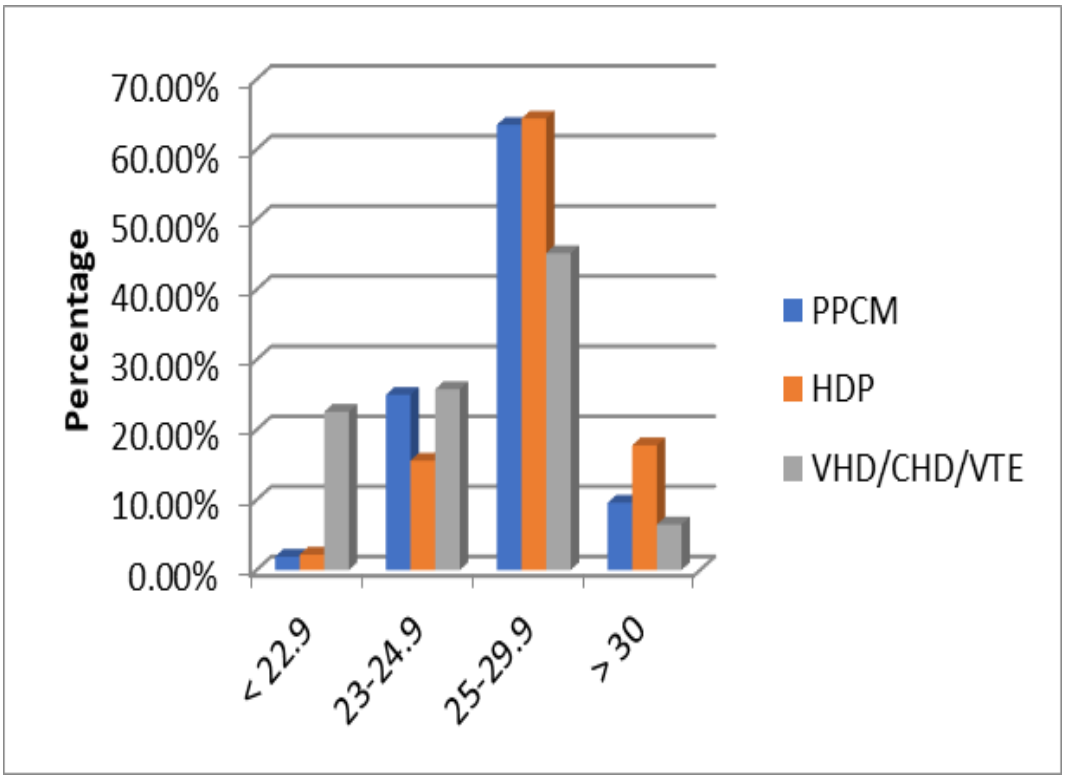

FIGURE 5: COMPARISON OF PATIENT PHENOTYPES ACCORDING TO BMI

TABLE 5: MEAN BMI ACROSS VARIOUS PATIENT PHENOTYPES

Mean BMI was highest (28.50) in VTE patients followed by HDP (27.31), PPCM (26.73) and VHD &CHD (25.00).

		N	Mean	Std. Deviation	Std. Error	Minimum	Maximum	F	p-value
BMI	PPCM	52	26.73	2.410	.334	22	32	4.893	0.001
	HDP	45	27.31	2.512	.374	22	32		
	CHD	9	25.00	1.871	.624	22	28		
	VHD	16	23.94	3.172	.793	20	32		
	VTE	6	28.50	6.892	2.814	24	42		
	OTHER	7	25.00	2.887	1.091	20	28		
	Total	135	26.47	3.039	.262	20	42		

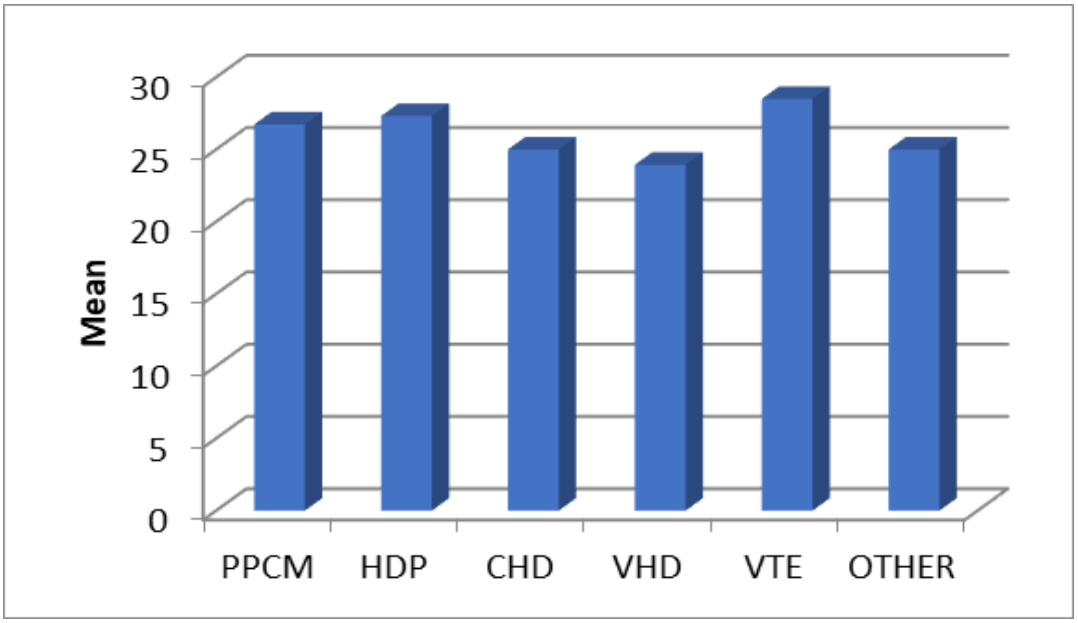

FIGURE 6: MEAN BMI ACROSS VARIOUS PATIENT PHENOTYPES

TABLE 6: TABLE SHOWING DISTRIBUTION OF PATIENT

BOOKED / UNBOOKED PREGNANCIES:

All patients were divided into two groups: those who were booked and un-booked. This classification was based on whether the patients had attended at least one antenatal clinic visit at our hospital. The results indicated a trend showing that more than two thirds of all the pregnancies were un-booked.

		PPCM		HDP		VHD/CHD/VTE		Total
		No. of cases	%age	No. of cases	%age	No. of cases	%age	
BOOKED/ UNBOOKED	BOOKED	15	28.8%	16	35.6%	11	35.5%	42
	UNBOOKED	37	71.2%	29	64.4%	20	64.5%	86
Total		52	100.0%	45	100.0%	31	100.0%	128

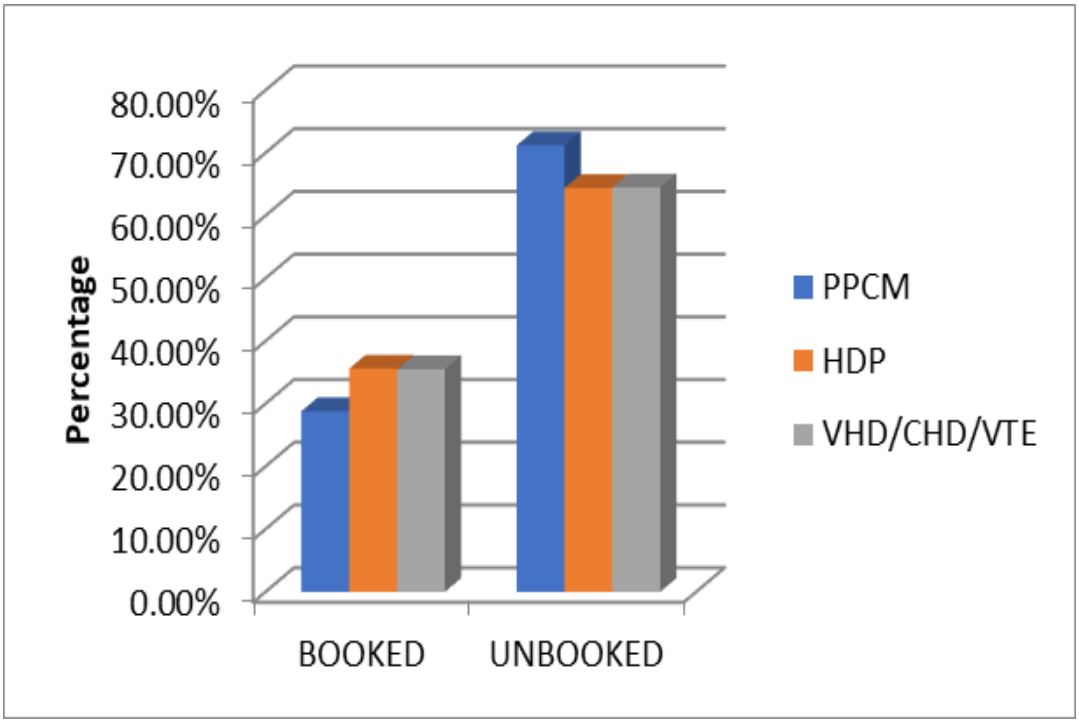

FIGURE 7: TABLE SHOWING DISTRIBUTION OF PATIENT

ADMITTED AT HEART CENTRE:

The table below presents data to evaluate the phenotypes of pregnant patients which required admission to the cardiology department. The results indicated that majority of patients admitted to the cardiac units were suffering from peripartum cardiomyopathy (PPCM) followed by venous thromboembolism (VTE), and congenital heart disease (CHD). In contrast patients diagnosed with HDP were least admitted in cardiology department which carries the future risk of cardiovascular diseases. These findings were statistically significant, with a p-value of 0.001.

TABLE 7: DISTRIBUTION OF PATIENTS ADMITTED IN HEART CENTRE

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
ADMITTED IN HEART CENTRE	NO	29	55.8%	44	97.8%	20	64.5%	93	22.793	0.001
	YES	23	44.2%	1	2.2%	11	35.5%	35		
Total		52	100.0%	45	100.0%	31	100.0%	128		

TABLE 8: DISTRIBUTION OF PATIENTS ADMISION ACCORDING TO ANTEPARTUM AND POST PARTUM STATUS

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
PRE LSCS/VD	NO	37	71.2%	43	95.6%	14	45.2%	94	24.131	0.001
	YES	15	28.8%	2	4.4%	17	54.8%	34		
POST LSCS/VD	NO	18	34.6%	2	4.4%	27	87.1%	47	54.131	0.001
	YES	34	65.4%	43	95.6%	4	12.9%	81		

The table above illustrates whether patients were enrolled prior to delivery, allowing for discussions on peripartum management between the cardiologist and obstetrician, or if they were transferred to the cardiac care unit after experiencing decompensation during the peripartum period. Patients with congenital heart disease (CHD), Valvular heart disease (VHD) and venous thromboembolism (VTE) were primarily enrolled before the planned lower segment cesarean section (LSCS), while patients with peripartum cardiomyopathy (PPCM) and hypertensive disorders of pregnancy (HDP) were enrolled after the LSCS. This suggests that patients with a known history of CHD, VHD or VTE were under regular cardiac follow-up prior to conception. They had undergone pre-pregnancy counseling and understood the risks associated with their current pregnancy. In contrast, patients with PPCM and HDP were referred to cardiac care during the peripartum period following decompensation.

TABLE 9: DISTRIBUTION OF PATIENTS ACCORDING TO CLINICAL PRESENTATION &NYHA CLASS

These 2 tables compared the clinical presentation of various phenotypes of heart disease and it showed that patients with PPCM and HDP had more severe form in symptoms in form of higher number of patients in NYHA Class IV and higher number of patients presenting in shock. (p value for both <0.05)

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
SHORTNESS OF BREATH	N	2	3.8%	43	95.6%	24	77.4%	69	90.759	0.001
	Y	50	96.2%	2	4.4%	7	22.6%	59		
PALPITATIONS	N	50	96.2%	45	100.0%	27	87.1%	122	6.979	0.031
	Y	2	3.8%	0	0.0%	4	12.9%	6		
FEVER	N	50	96.2%	44	97.8%	30	96.8%	124	0.212	0.900
	Y	2	3.8%	1	2.2%	1	3.2%	4		
POST PARTUM HEMORRHAGE	N	50	96.2%	42	93.3%	31	100.0%	123	2.174	0.337
	Y	2	3.8%	3	6.7%	0	0.0%	5		
SHOCK	N	46	88.5%	45	100.0%	30	96.8%	121	6.611	0.037
	Y	6	11.5%	0	0.0%	1	3.2%	7		

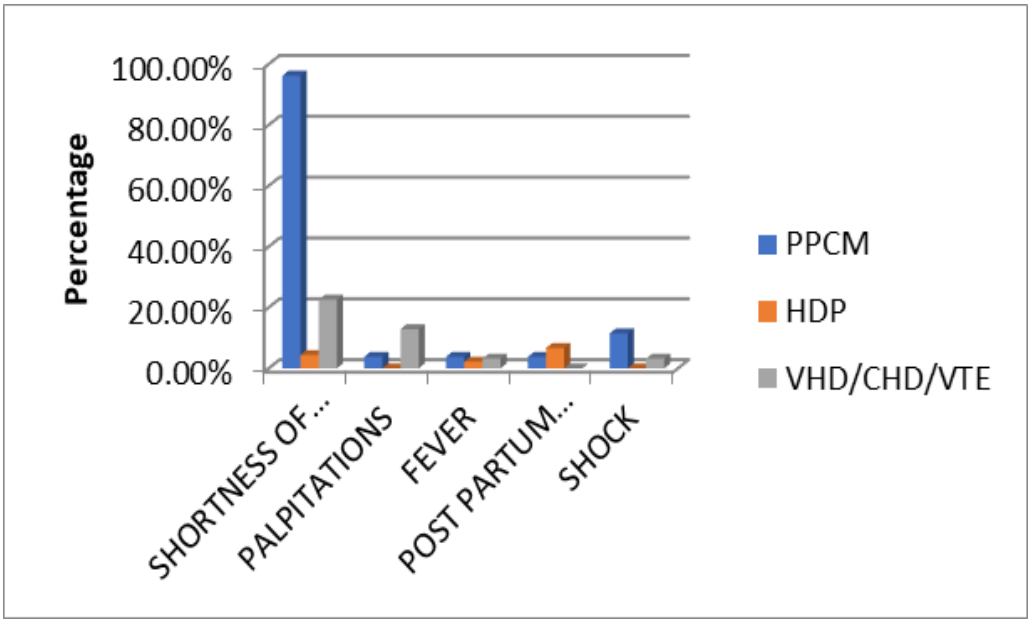

FIGURE 8: DISTRIBUTION OF PATIENTS ACCORDING TO CLINICAL PRESENTATION &NYHA CLASS

TABLE 10: DISTRIBUTION OF PATIENTS ACCORDING TO NYHA CLASS

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
NYHA CLASS	I	0	0.0%	0	0.0%	6	19.4%	6	115.94	0.001
	II	13	25.0%	2	4.4%	7	22.6%	22		
	III	20	38.5%	0	0.0%	1	3.2%	21		
	IV	17	32.7%	0	0.0%	1	3.2%	18		
	NA	2	3.8%	43	95.6%	16	51.6%	61		
Total		52	100.0%	45	100.0%	31	100.0%	128		

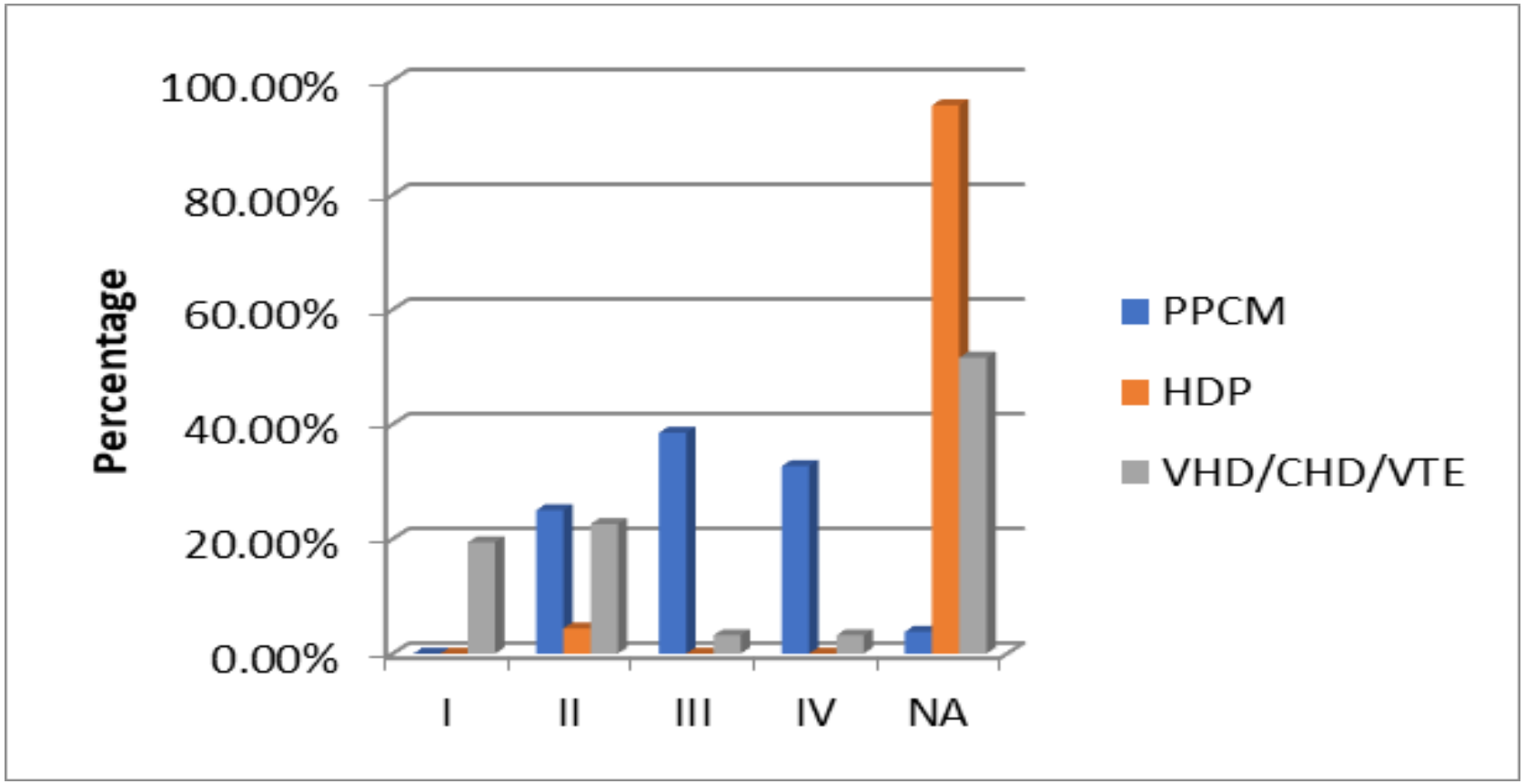

FIGURE 9: DISTRIBUTION OF PATIENTS ACCORDING TO NYHA CLASS

TABLE 11: MODE OF DELIVERY

Patients with VHD/CHD/VTE had more number of vaginal expulsion and curettage as compare to PPCM and HDP which was a significant association. It may be due to more number of medically contraindicated pregnancies in these patients.

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
Mode of Delivery	LSCS	40	76.9%	37	82.2%	20	64.5%	97	16.294	0.003
	VAGINAL DELIEVERY	12	23.1%	7	15.6%	5	16.1%	24		
	VAGINAL EXPULSION AND CHECK CURETTAGE	0	0.0%	1	2.2%	6	19.4%	7		

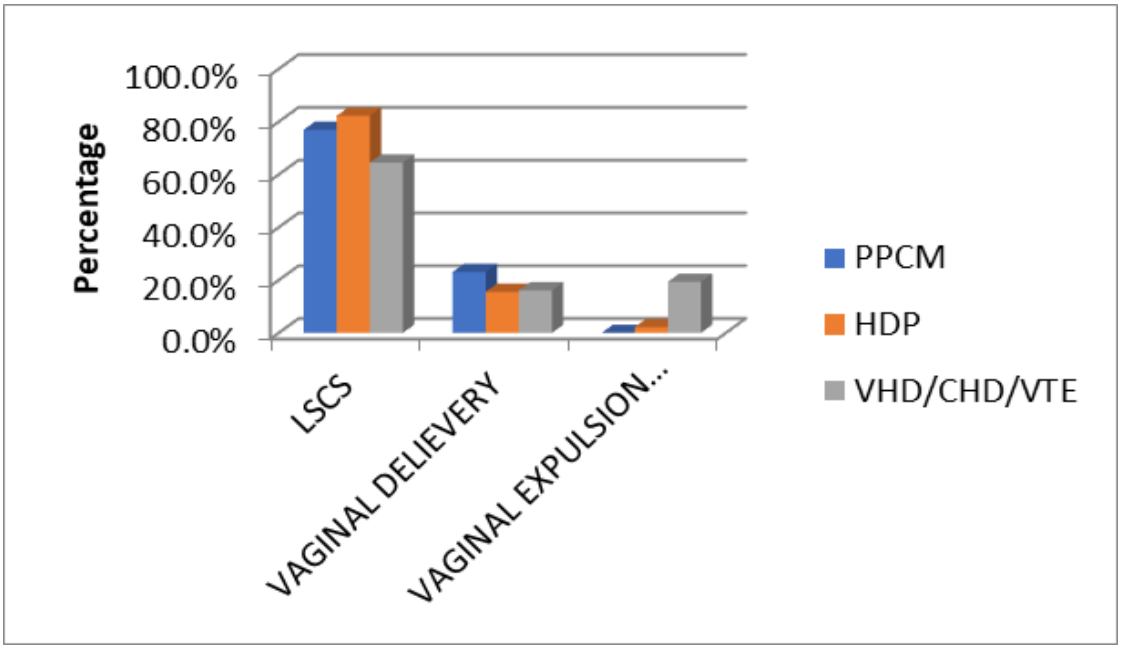

FIGURE 10: MODE OF DELIVERY

TABLE 12: TABLE SHOWING DIASTOLIC DYSFUNCTION AMONG PHENOTYPES

Patients with VHD/CHD/VTE were less likely to be associated with diastolic dysfunction as compared with PPCM and HDP patients.

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
DIASTOLIC DYSFUNCTION	NO	33	63.5%	28	62.2%	29	93.5%	90	10.598	0.005
	YES	19	36.5%	17	37.8%	2	6.5%	38		
Total		52	100.0%	45	100.0%	31	100.0%	128		

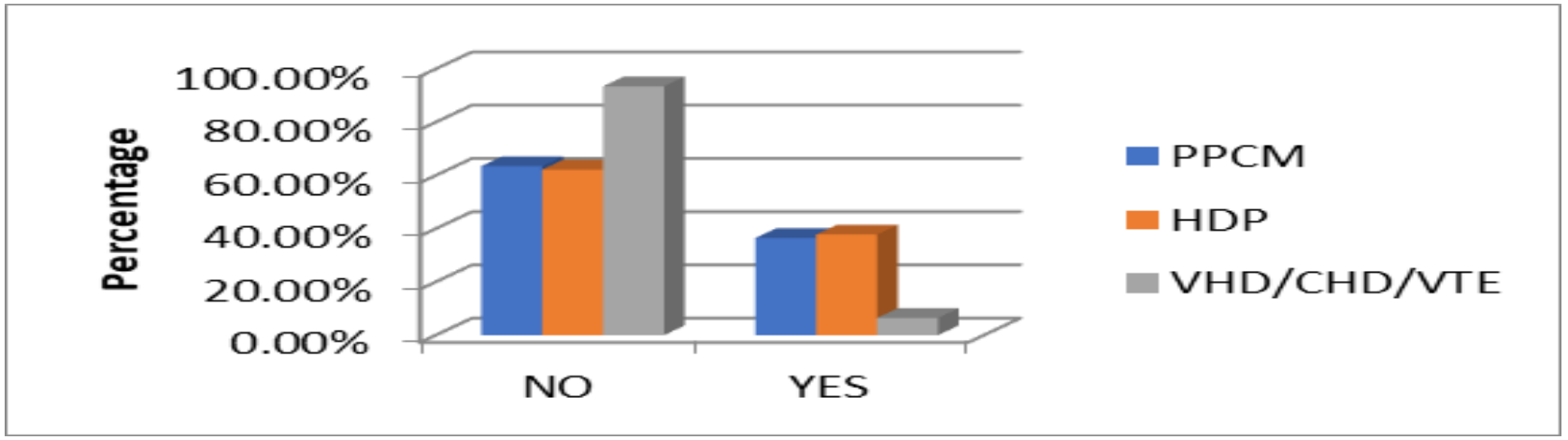

FIGURE 11: FIGURE SHOWING DIASTOLIC DYSFUNCTION AMONG PHENOTYPES

TABLE 13: MATERNAL OUTCOMES OF PATIENTS AMONG VARIOUS MEDICAL CONDITIONS

MATERNAL OUTCOMES:

Different phenotypes of patients were compared with their pregnancy outcomes and it showed that Patients with VHD, CHD or VTE had less rates of FGR as compared to PPCM and HDP patients. (p value 0.001). numerical trends showed lower number of pre term births in patients with VHD, CHD or VTE but this was not statistically significant finding.

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
FGR	N	49	94.2%	34	75.6%	31	100.0%	114	13.661	0.001
	Y	3	5.8%	11	24.4%	0	0.0%	14		
PPH	N	46	88.5%	42	93.3%	31	100.0%	119	3.970	0.137
	Y	6	11.5%	3	6.7%	0	0.0%	9		
PRE-TERM	N	21	40.4%	19	42.2%	20	64.5%	60	5.145	0.076
	Y	31	59.6%	26	57.8%	11	35.5%	68		

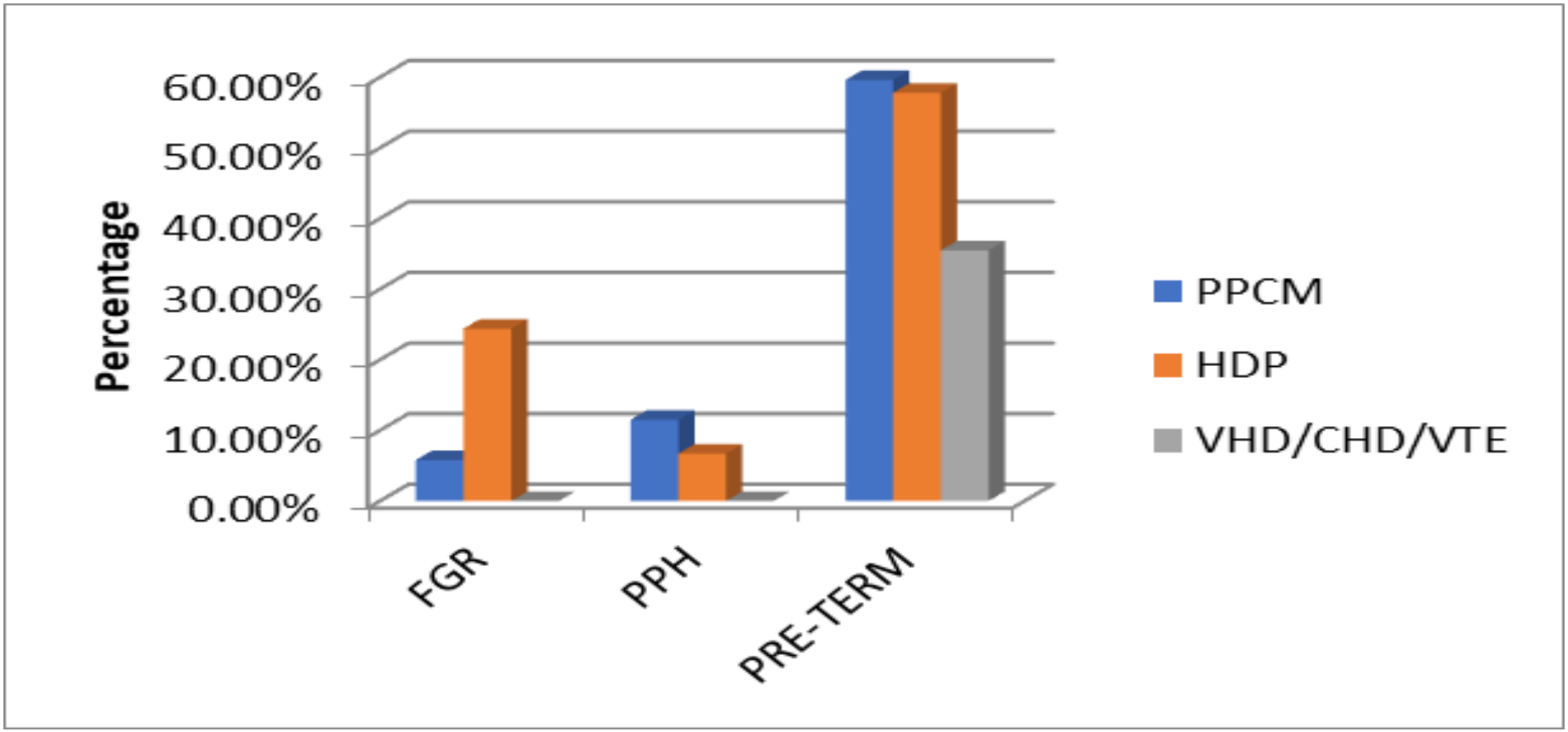

FIGURE 12: MATERNAL OUTCOMES OF PATIENTS AMONG VARIOUS MEDICAL CONDITIONS

TABLE 14: FETAL OUTCOMES IN PREGNANT FEMALES

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
FETAL/PERINATAL COMPLICATIONS	IUD	10	17.2%	6	11.5%	1	3.2%	17	34.622	0.001
	LOW BIRTH WEIGHT	17	29.3%	25	48.1%	4	12.9%	46		
	MISSED ABORTION	0	0.0%	0	0.0%	6	19.4%	6		
	NONE	31	53.4%	21	40.4%	20	64.5%	72		
Total		58	100.0%	52	100.0%	31	100.0%	141		

64.5% of patients with VHD, CHD or VTE were free from any fetal complications which showed significant difference when compared with PPCM and HDP patients (53.4% and 40.4% respectively).

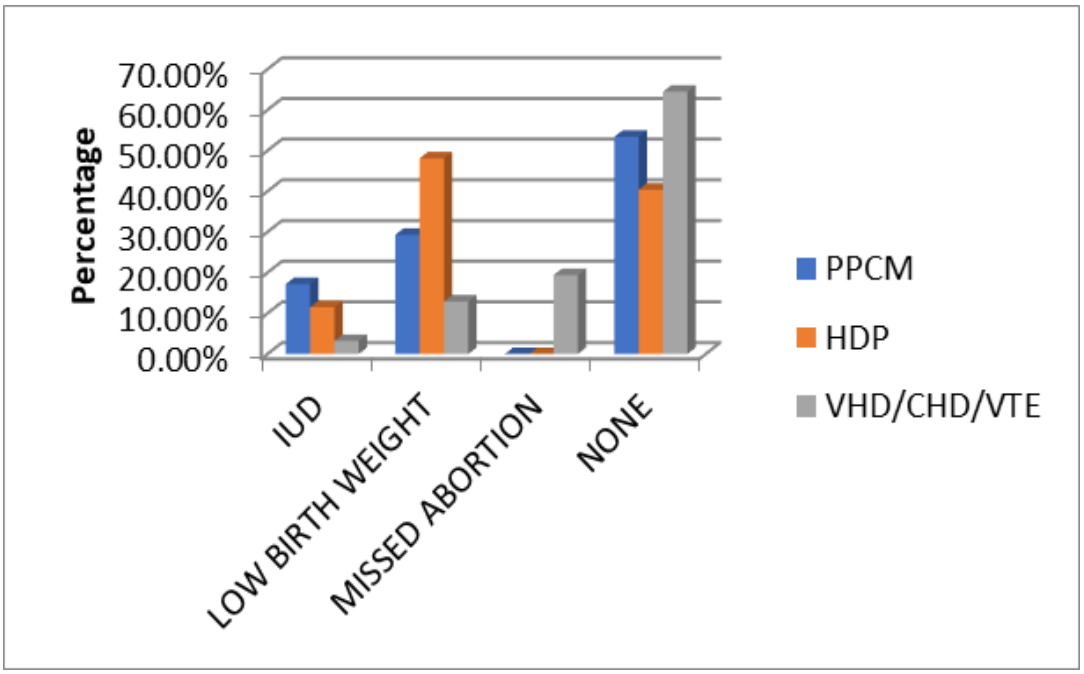

FIGURE 13: FETAL OUTCOMES IN PREGNANT FEMALES

TABLE 15: NEED FOR MECHANICAL VENTILATOR IN PREGNANT FEMALE

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
MECHANICAL VENTILATOR	N	43	82.7%	44	97.8%	30	96.8%	117	8.489	0.014
	Y	9	17.3%	1	2.2%	1	3.2%	11		
Total		52	100.0%	45	100.0%	31	100.0%	128		

A patient with PPCM was strongly associated with need for mechanical ventilation vis-à-vis HDP and VHD/CHD/VTE.

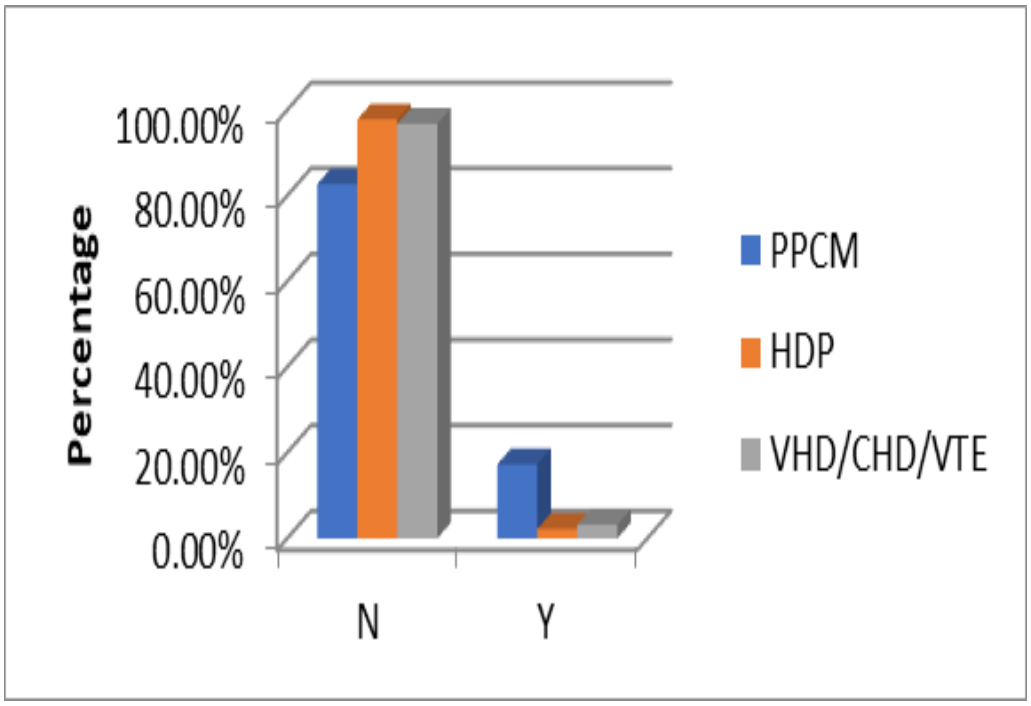

FIGURE 14: NEED FOR MECHANICAL VENTILATOR IN PREGNANT FEMALE

TABLE 16: MORTALITY DATA IN PREGNANT FEMALES

		PPCM		HDP		VHD/CHD/VTE		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age	No. of cases	%age			
MORATILTY	N	42	80.8%	44	97.8%	31	100.0%	117	12.73	0.002
	Y	10	19.2%	1	2.2%	0	0.0%	11		
Total		52	100.0%	45	100.0%	31	100.0%	128		

Patients with PPCM had significantly more mortality rate as compared with HDP and VHD/CHD/VTE patients.

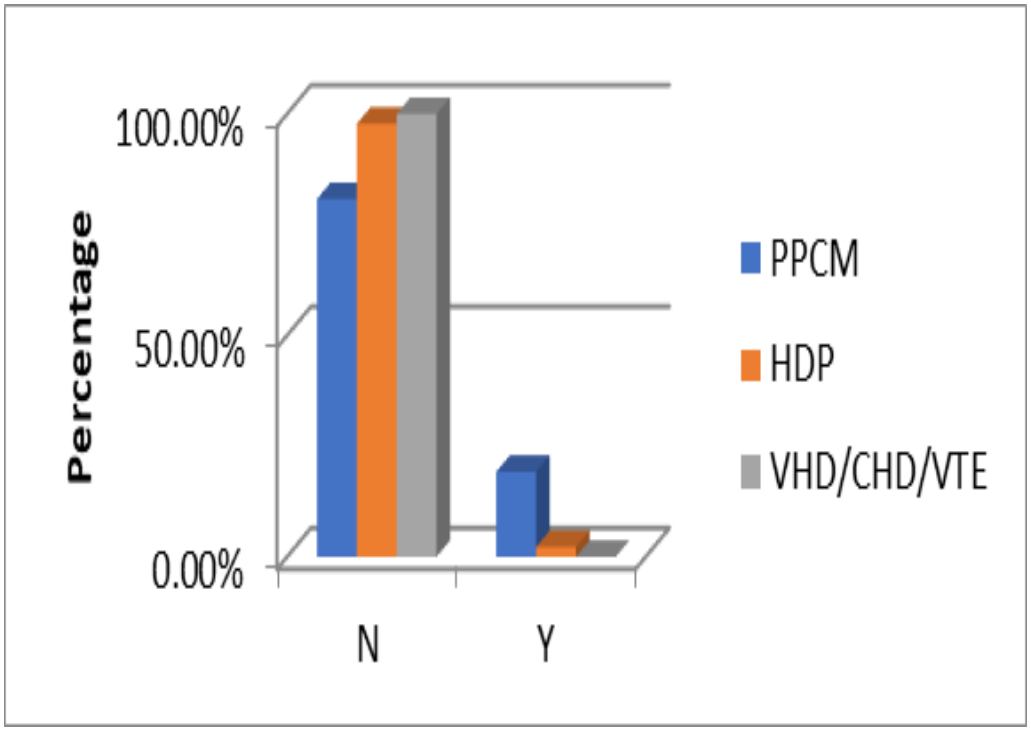

FIGURE 15: MORTALITY DATA IN PREGNANT FEMALES

TABLE 17: DISTIBUTION OF PERIPARTUM CARDIOMYOPATHY PATIENTS ACCODING TO VARIOUS AGE GROUPS

		PPCM	
		No. of cases	%age
AGE GROUP	< 25	7	13.5%
	26-30	20	38.5%
	31-35	16	30.8%
	36-40	6	11.5%
	> 40	3	5.8%
Total		52	100.0%

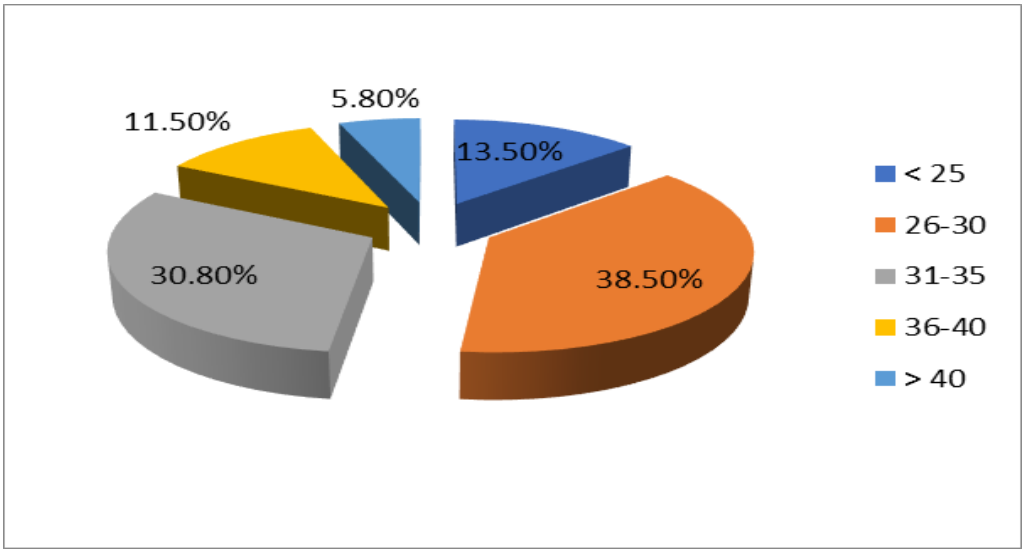

FIGURE 16: DISTIBUTION OF PERIPARTUM CARDIOMYOPATHY PATIENTS ACCODING TO VARIOUS AGE GROUPS

Mean age and gestational period of the patients with PPCM was 31.10+-5.02 years and 34.02+-4.58 weeks respectively.

TABLE 18: NUMBER OF PATIENTS DIAGNOSED IN POST PARTUM

	DIAGNOSIS	PPCM	
		No. of cases	%age
POST PARTUM DIAGNOSIS	N	24	43.6%
	Y	31	56.4%
Total		52	100.0%

56.4% Patients had symptoms or were diagnosed to have PPCM in postnatal period while rest were diagnosed in antenatal period.

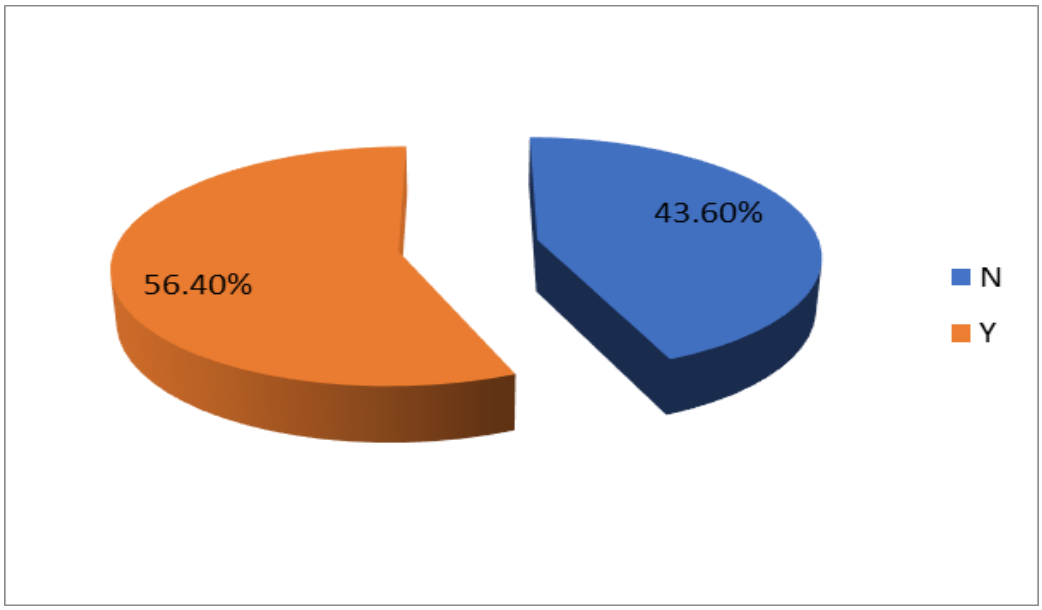

FIGURE 17: NUMBER OF PATIENTS DIAGNOSED IN POST PARTUM

TABLE 19: DISTRIBUTION OF PPCM PATIENTS ACCORDING TO PARITY

DIAGNOSIS	PPCM	
	No. of cases	%age
MULTI	24	43.6%
PRIMI	31	56.4%

Among patients with PPCM, 56.4% were primiparous and rest 43.6% were multiparous.

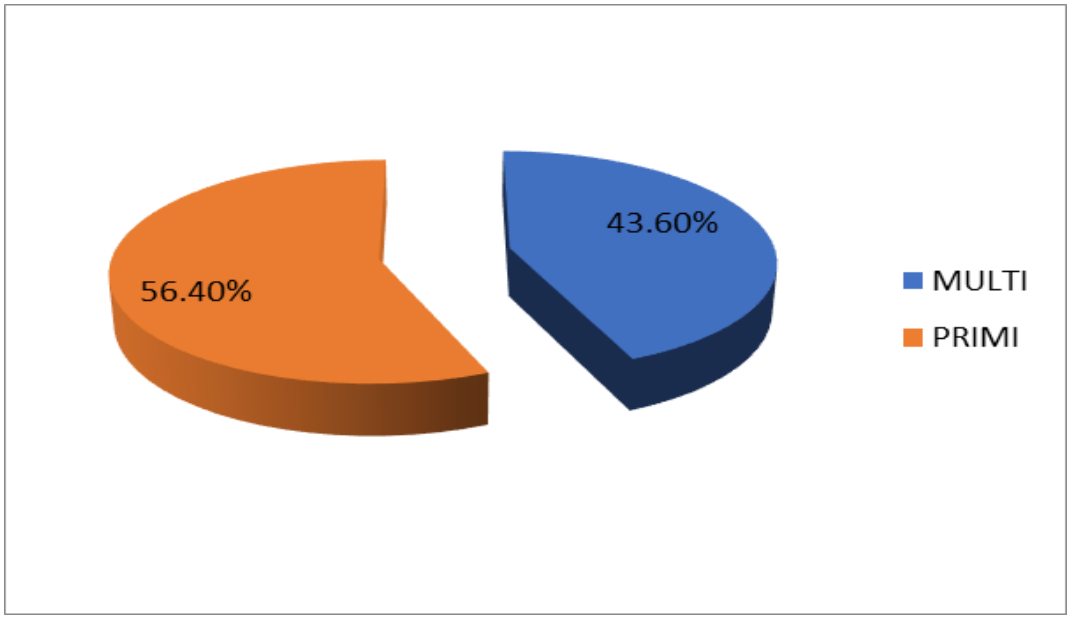

FIGURE 18: DISTRIBUTION OF PPCM PATIENTS ACCORDING TO PARITY

TABLE 20: DISTRIBUTION OF PATIENTS ACCORDING TO BMI

		PPCM	
		No. of cases	%age
BMI	< 22.9	1	1.9%
	23-24.9	13	25.0%
	25-29.9	33	63.5%
	> 30	5	9.6%
Total		52	100.0%

73.1 % patients had BMI > 25kg/m2 which means obesity was prevalent.

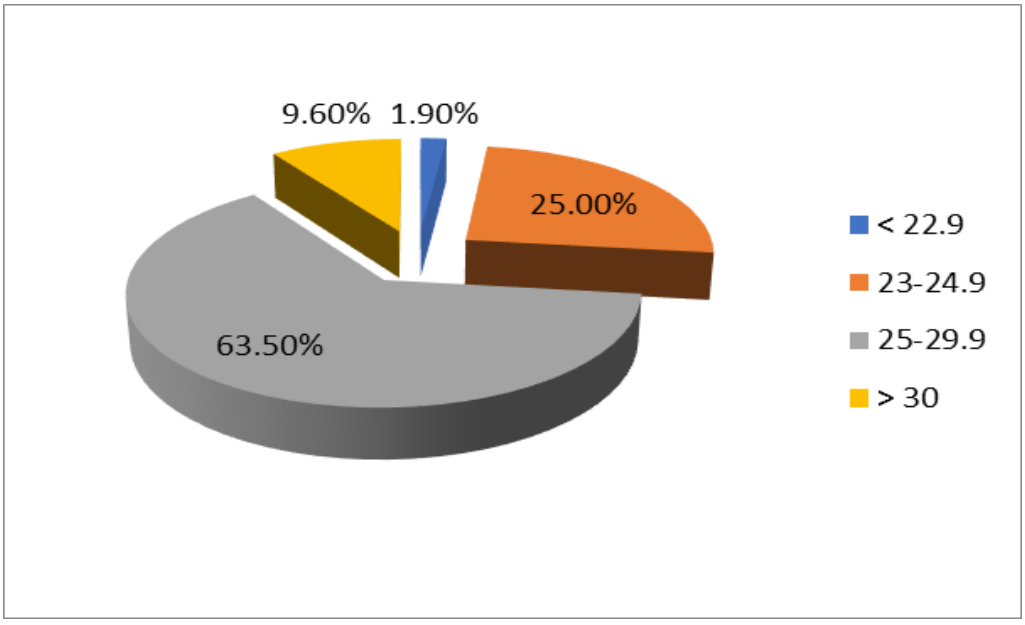

FIGURE 19: DISTRIBUTION OF PATIENTS ACCORDING TO BMI

Out of 52 patients; 6 patients had twin pregnancies among patients who had PPCM; 10 patients had history of abortions in the past who had PPCM; and 2 patients had history of IVF treatment in current pregnancy.

76.9% Patients delivered via LSCS and remaining via vaginal delivery.

TABLE 21: CHIEF COMPLAINTS OF PPCM PATIENTS

		PPCM	
		No. of cases	%age
SHORTNESS OF BREATH	N	2	3.8%
	Y	50	96.2%
PALPITATIONS	N	50	96.2%
	Y	2	3.8%
GTCS	N	51	98.1%
	Y	1	1.9%
INCREASED BP READINGS	N	30	57.7%
	Y	22	42.3%
FEVER	N	50	96.2%
	Y	2	3.8%
POST PARTUM HEMORRHAGE	N	50	96.2%
	Y	2	3.8%
SHOCK	N	46	88.5%
	Y	6	11.5%
OTHERS	CHEST PAIN	1	1.9%
	PRESYNCOPE	1	1.9%

Most common presenting complaint was shortness of breath (96.2%), followed by increased BP readings IN 42.3% of patients, and 11.5% patients presented in shock with need for inotropes.

TABLE 22: NYHA CLASS AT PRESENTATION OF PPCM PATIENTS

		PPCM	
		No. of cases	%age
NYHA CLASS(ADMSN)	I	0	0.0%
	II	13	25.0%
	III	20	38.5%
	IV	17	32.7%
	NA	2	3.8%
Total		52	100.0%

The most common NYHA classes at admission for PPCM patients were Class III (38.5%) and Class IV (32.7%), indicating that a large proportion of patients had severe limitations in their physical activity or symptoms at rest. Class II was seen in 25.0% of cases.2 patients were not assigned NYHA class because 1 presented with eclampsia and other was asymptomatic. This distribution suggests that most patients presented with moderate to severe heart failure symptoms at the time of admission.

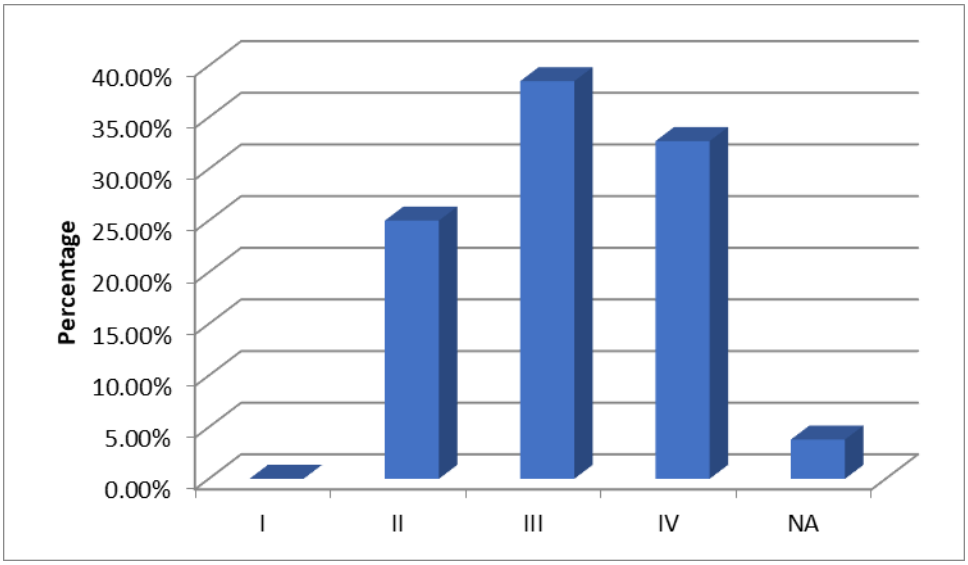

FIGURE 20: NYHA CLASS AT PRESENTATION OF PPCM PATIENTS

TABLE 23: DISTRIBUTIONS OF VARIOUS COMORBIDITIES IN PPCM PATIENTS

	DIAGNOSIS	PPCM	
		No. of cases	%age
HYPERTENSION	N	40	76.9%
	Y	12	23.1%
BRONCHIAL ASTHMA	N	51	98.1%
	Y	1	1.9%
HYPOTHYROIDISM	N	44	84.6%
	Y	8	15.4%
HYPERTHYROIDISM	N	51	98.1%
	Y	1	1.9%
DM	N	49	94.2%
	Y	3	5.8%

This table presents the distribution of various comorbidities among PPCM patients and it showed that Hypertension was present in 23.1% patients while other conditions like Bronchial asthma, hyperthyroidism, and diabetes mellitus (DM) were relatively rare, affecting only 1.9%, 1.9%, and 5.8% of patients, respectively. Hypothyroidism was present in 15.4% of the patients.

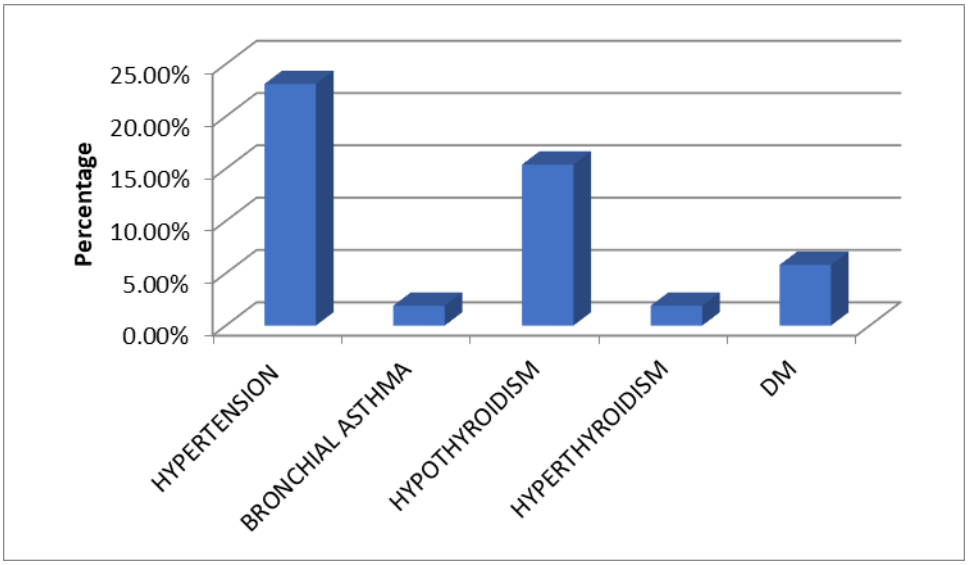

FIGURE 22: DISTRIBUTIONS OF VARIOUS COMORBIDITIES IN PPCM PATIENTS

TABLE 24: BASELINE INVESTIGATIONS IN PPCM PATIENTS

	PPCM	
	Mean	SD
HEMOGLOBIN (g/dL)	11.00	2.17
BNP	1168.81	1057.61
QRS (ms)	103.67	13.65
EF (%)	31.13	10.59
RVFAC	32.25	7.11
PASP (mmHg)	34.44	12.70
LVEDD (mm)	54.17	5.79
LVESD (mm)	38.06	6.54

Mean hemoglobin levels were 11.00+-2.17 gm/dl.

Mean BNP levels were 1168.81+-1057.61

The mean ejection fraction at baseline in these patients was 31.13%+-10.59.

Mean LVEDD and LVESD were 54.17+-5.79 mm and 38.06+-6.54 mm respectively.

TABLE 25: VARIOUS ASSOCIATIONS WITH PPCM

		PPCM	
		No. of cases	%age
GDMA2	N	48	92.3%
	Y	4	7.7%
PRE-ECLAMPSIA	N	39	75.0%
	Y	13	25.0%
PRE-ECLAMPSIA WITH SEVERE FEATURES	N	44	84.6%
	Y	8	15.4%
ECLAMPSIA	N	51	98.1%
	Y	1	1.9%
SEPSIS	N	43	82.7%
	Y	9	17.3%
AKI	N	51	98.1%
	Y	1	1.9%
OTHERS	PRES	1	1.9%

This table presents data on various clinical conditions and complications associated with Peripartum Cardiomyopathy (PPCM) and it showed that 42.3% of patients with PPCM were associated with PIH (21 patients had preeclampsia and 1 patient had eclampsia) while 7.7% of patients had GDM. 9 patients (17.3%) were associated with sepsis and 1 patient had acute kidney injury.

TABLE 26: MATERNAL OUTCOMES IN PPCM PATIENTS

		PPCM	
		No. of cases	%age
PRIMARY OUTCOMES	NONE	1	1.9%
	BIVENTRICULAR FAILURE	1	1.9%
	LEFT VENTRICULAR FAILURE	41	78.8%
	MATERNAL DEATH	9	17.3%
Total		52	100.0%

Left ventricular failure was observed in 78.8%, biventricular failure was less prevalent and affected 1.9% of patients, suggesting that a small subset of patients experienced progressive cardiac compromise. There was only one patient with H/O PPCM in a previous pregnancy who remained asymptomatic during the current pregnancy and did not experience any primary outcomes. Additionally, 9 patients out of 52 needed mechanical ventilatory support in the peripartum period.

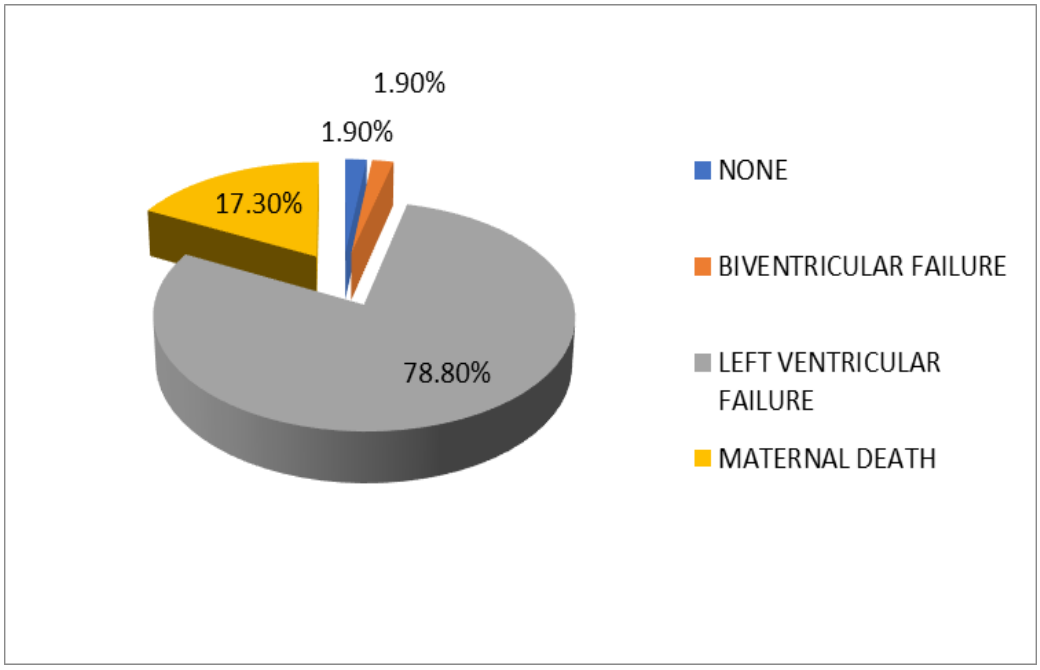

FIGURE 22: MATERNAL OUTCOMES IN PPCM PATIENTS

TABLE 27: OBSTETRIC OUTCOMES AMONG PPCM PATIENTS

		PPCM	
		No. of cases	%age
FGR	N	49	94.2%
	Y	3	5.8%
ANHYDROAMNIOS	N	51	98.1%
	Y	1	1.9%
OLIGOHYDROAMNIOS	N	51	98.1%
	Y	1	1.9%
POLYHYDRAMNIOS	N	51	98.1%
	Y	1	1.9%
PPH	N	46	88.5%
	Y	6	11.5%
PRE-TERM	N	21	40.4%
	Y	31	59.6%
PPROM	N	51	98.1%
	Y	1	1.9%

A significant number of patients with PPCM had preterm deliveries i.e. 59.6% while PPH was seen in 11.5% patients.

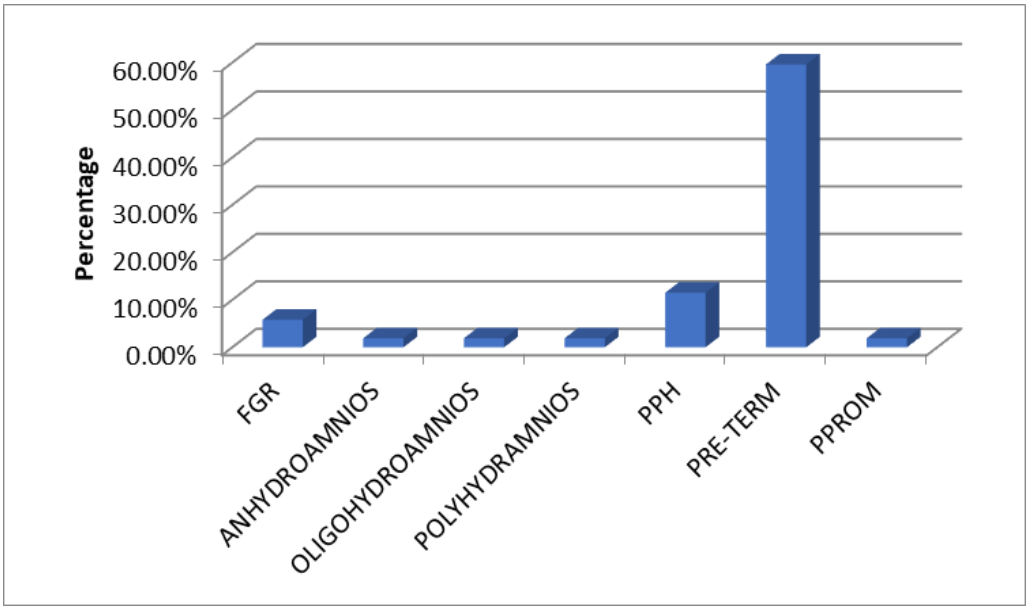

FIGURE 23: OBSTETRIC OUTCOMES AMONG PPCM PATIENTS

TABLE 28: FETAL OUTCOMES AMONG PPCM PATIENTS

		PPCM	
		No. of cases	%age
FETAL/PERINATAL COMPLICATIONS	IUD	10	17.2%
	LOW BIRTH WEIGHT	17	29.3%
	NONE	31	53.4%
Total		58	100.0%

In patients with Peripartum Cardiomyopathy (PPCM), fetal and perinatal complications were notably high, with low birth weight and intrauterine demise (IUD) as the primary concerns. Among the 58 fetal outcomes (adjusted to account for six twin pregnancies), low birth weight was observed in 29.3% of cases, while IUD accounted for 17.2% of cases. This significant prevalence of complications reflects the critical impact of PPCM on fetal health, contributing to adverse outcomes that require careful monitoring.

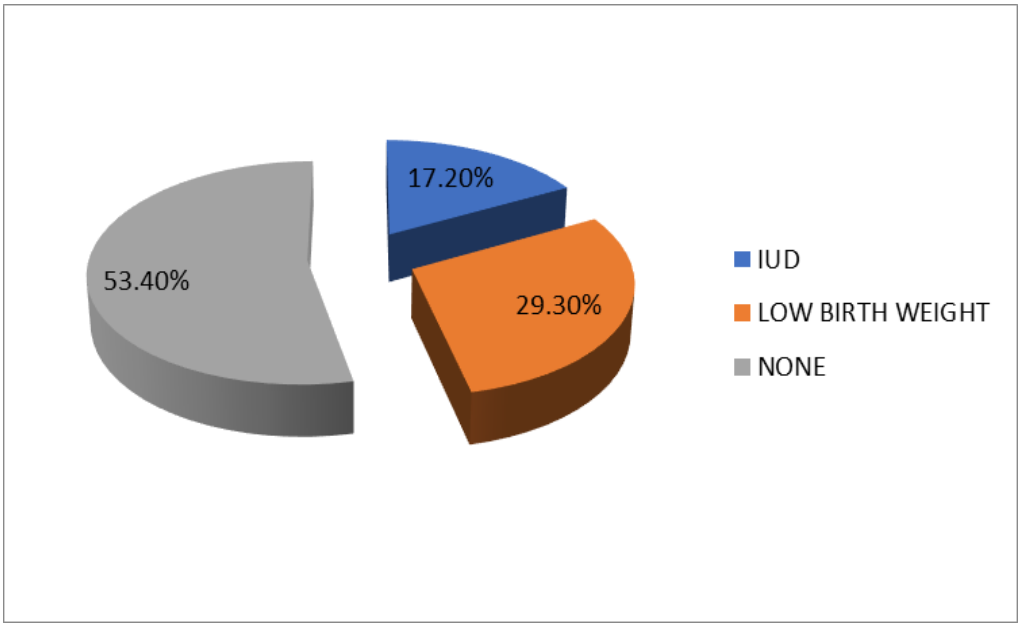

FIGURE 24: FETAL OUTCOMES AMONG PPCM PATIENTS

TABLE 29: DISCHARGE MEDICATIONS AMONG PPCM PATIENTS

		PPCM	
		No. of cases	%age
BETA BLOCKERS	N	21	40.4%
	Y	31	59.6%
RAAS #	N	30	57.7%
	ACE-I	10	19.2%
	ARB	7	13.5%
	ARNI	5	9.6%
LOOP DIURETICS	N	42	80.8%
	Y	10	19.2%
MRA	N	29	55.8%
	Y	23	44.2%
ANTICOAGULATION	N	51	98.1%
	NOAC	0	0.0%
	VKA	1	1.9%
OTHERS	SGLT2#	3	5.8%
	INSULIN	2	3.8%
	HYDRALAZINE	1	1.9%
	IVABRADINE	4	7.7%
Total		52	100.0%

Most patients (59.6%) were prescribed beta blockers to control heart rate and reduce oxygen demand, while 42.3% were on RAAS inhibitors (ACE-I, ARB, or ARNI) to aid fluid balance and lower vascular resistance. Loop diuretics were used in 19.2% to address fluid overload, and MRAs were prescribed to 44.2% to prevent excess fluid retention by inhibiting aldosterone. Only 1.9% of patients required anticoagulation with warfarin, likely due to either a low thromboembolic risk or careful risk management. A small number of patients used medications like SGLT2 inhibitors, ivabradine, and hydralazine. This data gives insight into the therapeutic management of PPCM, indicating that beta blockers and mineralocorticoid receptor antagonists were commonly used in these patients.

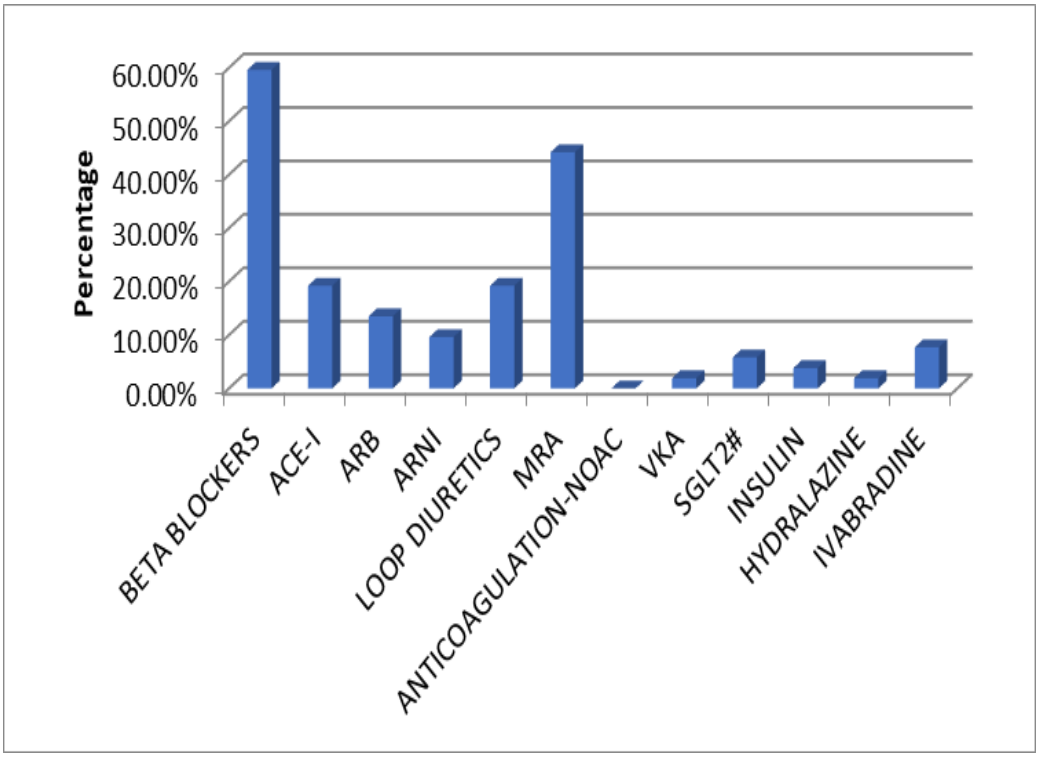

FIGURE 25: DISCHARGE MEDICATIONS AMONG PPCM PATIENTS

TABLE 30: RECOVERY PREDICTORS AMONG PPCM PATIENTS

		RECOVERED				Total	Chi-square value	p-value
		NO		YES				
AGE GROUP	< 25	4	22.2%	3	12.5%	7	1.857	0.762
	26-30	7	38.9%	8	33.3%	15		
	31-35	5	27.8%	7	29.2%	12		
	36-40	1	5.6%	4	16.7%	5		
	> 40	1	5.6%	2	8.3%	3		
BOOKED/ UNBOOKED	BOOKED	6	33.3%	8	33.3%	14	0.000	1.000
	UNBOOKED	12	66.7%	16	66.7%	28		
POST PARTUM GP	N	6	33.3%	8	33.3%	14	0.000	1.000
	Y	12	66.7%	16	66.7%	28		
Parity	MULTI	8	44.4%	11	45.8%	19	0.008	0.929
	PRIMI	10	55.6%	13	54.2%	23		

It was defined as LVEF>50% at 6 months of follow-up in our study.

42 out of 52 PPCM patients underwent this analysis of recovery, out of which 24 patients had complete recovery at 6 months (57.14%) and rest were categorised as unrecovered patients.

These patients were evaluated to learn the recovery predictors in our PPCM group of patients

PATIENT CHARACTERISTICS:

This table shows a comparison between recovered and non-recovered cases of Peripartum Cardiomyopathy (PPCM) based on several variables (age group, booked pregnancy or not, diagnosed PPCM antepartum or postpartum, and parity). 61.1% of non-recovered patients were younger (age<30 years) as compared to recovered patients (45.8%). Also, the mean age in recovered group was higher than non-recovered group (32.13+-5.16 vs 29.83+-5.45 years) but p value was not significant (p value=0.172). So, this can only be a trend seen in our results as these were not statistically significant. None of other variables showed a statistically significant difference between recovered and non-recovered patients with PPCM.

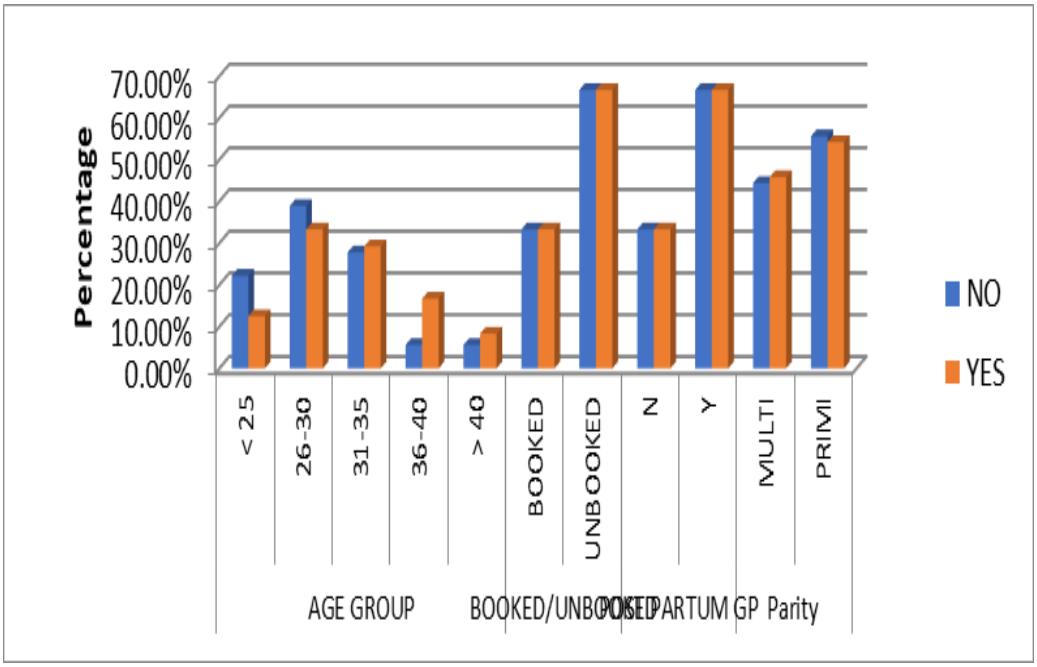

FIGURE 26: RECOVERY PREDICTORS AMONG PPCM PATIENTS

TABLE 31: BASELINE CHARACTERISTICS OF RECOVERED/UN RECOVERED PPCM PATIENTS

RECOVERED	NO		YES		Z	p-value
	Mean	SD	Mean	SD		
Mean Age	29.83	5.45	32.13	5.16	-1.391	0.172
BMI	26.22	2.24	26.96	2.60	-0.964	0.341
PREGNANCY DURATION AT TERMINATION	33.88	3.06	35.98	1.64	-2.855	0.007

The table above shows that mean BMI of both the groups was comparable and it was not a recovery predictor in our study.

Period of gestation was higher in recovered group as compared to non-recovered group (35.98+-1.64 vs 33.88+-3.06 weeks) which was also statistically significant; this finding may be due to grave clinical presentation (higher NYHA class) in non-recovered patients with PPCM which might have led to early termination of pregnancy in these patients due to deteorating maternal condition.

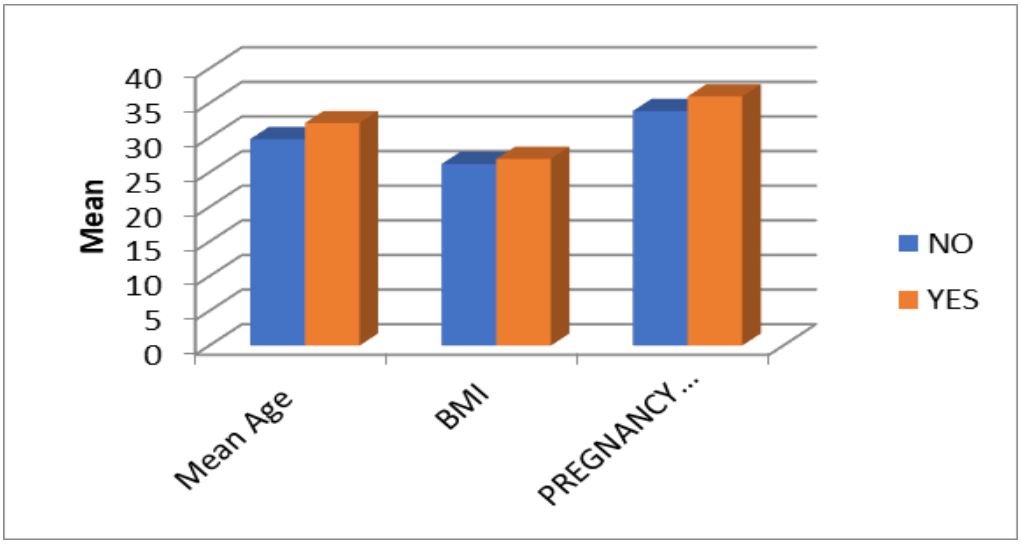

FIGURE 27: BASELINE CHARACTERISTICS OF RECOVERED/UN RECOVERED PPCM PATIENTS

TABLE 32: DISTRIBUTION OF NYHA CLASS ACCORDING TO RECOVERY

		RECOVERED				Total	Chi-square value	p-value
		NO		YES				
NYHA CLASS	II	4	22.2%	9	37.5%	13	10.76	0.016
	III	5	27.8%	13	54.2%	18		
	IV	8	44.4%	1	4.2%	9		
	NA	1	5.6%	1	4.2%	2		

This table shows the comparison between recovered and non-recovered cases of Peripartum Cardiomyopathy (PPCM) based on NYHA functional class at admission, along with the Chi-square value and p-value to assess the statistical significance. Results showed that NYHA class IV at admission, indicating worse heart failure symptoms, is associated with a higher likelihood of non-recovery. For example, 44.4% of non-recovered cases were in Class IV, while only 4.2% of recovered patients were in NYHA Class IV at admission. On the other hand, patients in Class I-III have a higher probability of recovery. This data suggests that the severity of heart failure at the time of admission (as measured by NYHA class) has a significant impact on the likelihood of recovery in PPCM patients.

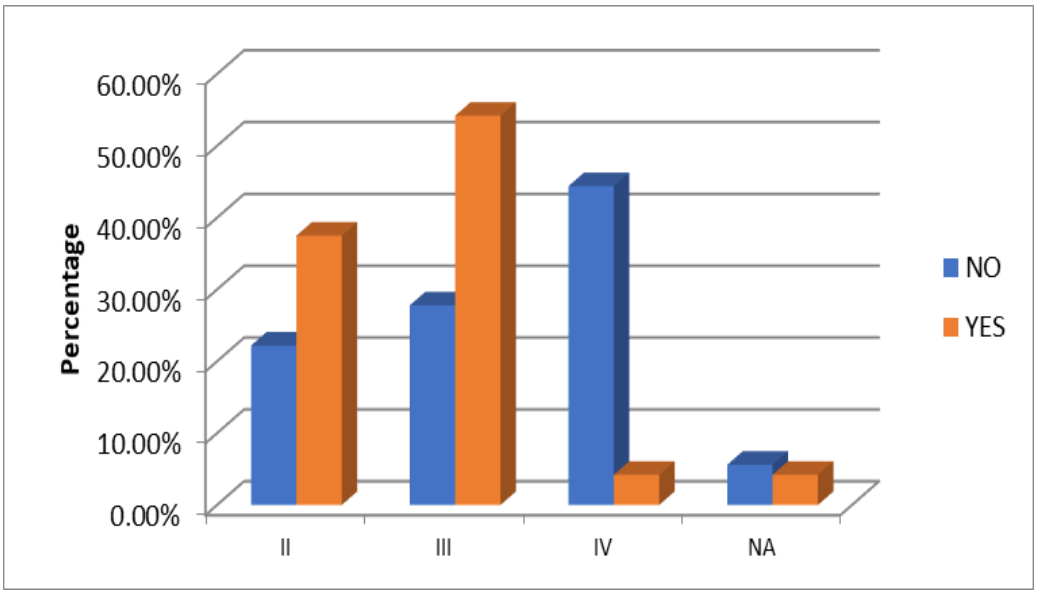

FIGURE 28: DISTRIBUTION OF NYHA CLASS ACCORDING TO RECOVERY

TABLE 33: LABORATORY PARAMETERS IN RECOVERED/ UNRECOVERED PATIENTS

	NO		YES			p-value
	Mean	SD	Mean	SD	Z	
HEM OGLOBIN	11.37	1.93	11.36	1.86	0.024	0.981
BNP	1623.89	1343.53	584.92	374.38	3.619	0.001

The mean BNP levels were significantly higher in non-recovered patients (1623.89 pg/mL) compared to recovered patients (584.92 pg/mL), with a p-value of 0.001, indicating a strong association between higher BNP levels and non-recovery. BNP is a marker of cardiac stress and heart failure severity, so this result suggests that higher BNP levels at diagnosis are associated with worse outcomes in PPCM patients. Thus, it was concluded that haemoglobin levels do not show a significant difference between the recovery and non-recovery groups while BNP levels were significantly higher in non-recovered PPCM patients, indicating that elevated BNP may be a predictive marker of poor recovery in this condition.

TABLE 34: ECHOCARDIOGRAPHIC PARAMETERS IN RECOVERED/ UNRECOVERED PATIENTS

	NO		YES			
	Mean	SD	Mean	SD	Z	p-value
EF (%)	27.94	9.82	36.67	8.83	-3.019	0.004
PASP	39.00	11.64	27.46	10.70	3.332	0.002
LVEDD	57.11	5.21	51.42	5.26	3.486	0.001

This table shows comparison of key cardiac metrics—EF, PASP, and LVEDD—between recovered and non-recovered PPCM patients highlighting the importance of these indicators in predicting recovery outcomes. Non-recovered patients showed a significantly lower mean EF of 27.94% compared to 36.67% in recovered patients (p = 0.004), suggesting that an initially lower EF may predict poorer recovery. Additionally, PASP was significantly elevated in non-recovered patients, with a mean of 39.00 mmHg versus 27.46 mmHg in those who recovered (p = 0.002), linking increased PASP with adverse outcomes. LVEDD was also notably larger in non-recovered patients, averaging 57.11 mm compared to 51.42 mm in recovered patients (p = 0.001), indicating that increased ventricular dilation correlates with a higher likelihood of non-recovery. These findings collectively suggest that lower EF, elevated PASP, and larger LVEDD at diagnosis may serve as crucial markers for identifying PPCM patients at risk of non-recovery, offering valuable insights for clinical management and intervention strategies

Other echocardiographic parameters like presence/grading of diastolic dysfunction and presence of RV dysfunction did not show any corelation with the recovery in our results.

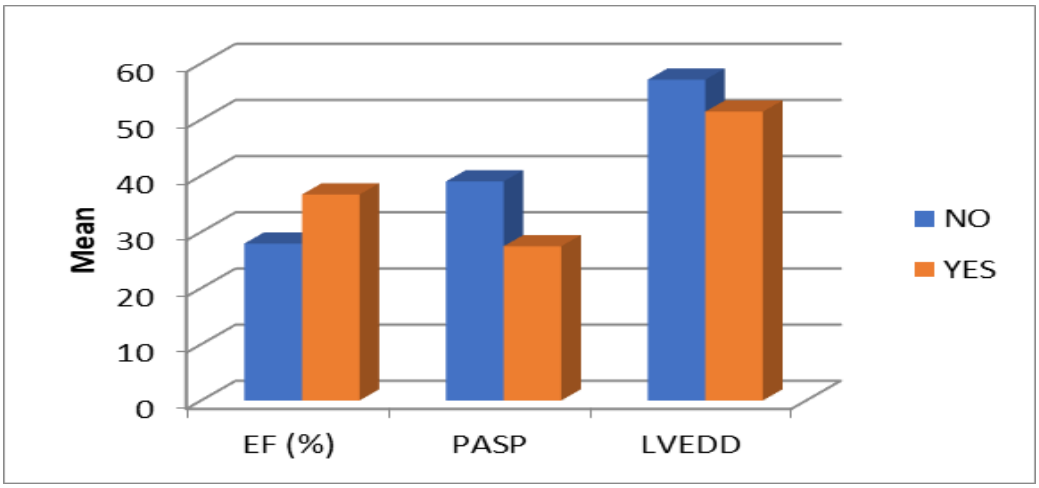

FIGURE 29: ECHOCARDIOGRAPHIC PARAMETERS IN RECOVERED/ UNRECOVERED PATIENTS

TABLE 35: TRENDS OF DISCHARGE MEDICATIONS IN RECOVERED/ UNRECOVERED PATIENTS

		RECOVERED				Total	Chi-square value	p-value
		NO		YES				
BETA BLOCKERS	N	6	33.3%	5	20.8%	11	0.831	0.483
	Y	12	66.7%	19	79.2%	31		
RAAS #	N	11	61.1%	9	37.5%	20	6.213	0.102
	ACE-I	3	16.7%	7	29.2%	10		
	ARB	4	22.2%	3	12.5%	7		
	ARNI	0	0.0%	5	20.8%	5		
LOOP DIURETICS	N	11	61.1%	21	87.5%	32	3.948	0.070
	Y	7	38.9%	3	12.5%	10		
MRA	N	6	33.3%	13	54.2%	19	1.802	0.221
	Y	12	66.7%	11	45.8%	23		
OTHERS	HYDRALAZINE	1	5.6%	0	0.0%	1	3.676	0.452
	INSULIN	0	0.0%	2	8.3%	2		
	IVABRADINE	1	5.6%	3	12.5%	4		
	SGLT2#	1	5.6%	2	8.3%	3		

Recovered patients showed more use of beta blockers, ARNI's, and ivabradine while less use of loop diuretics was observed in these patients. This was only a trend seen in our results and none of these were statistically significant.

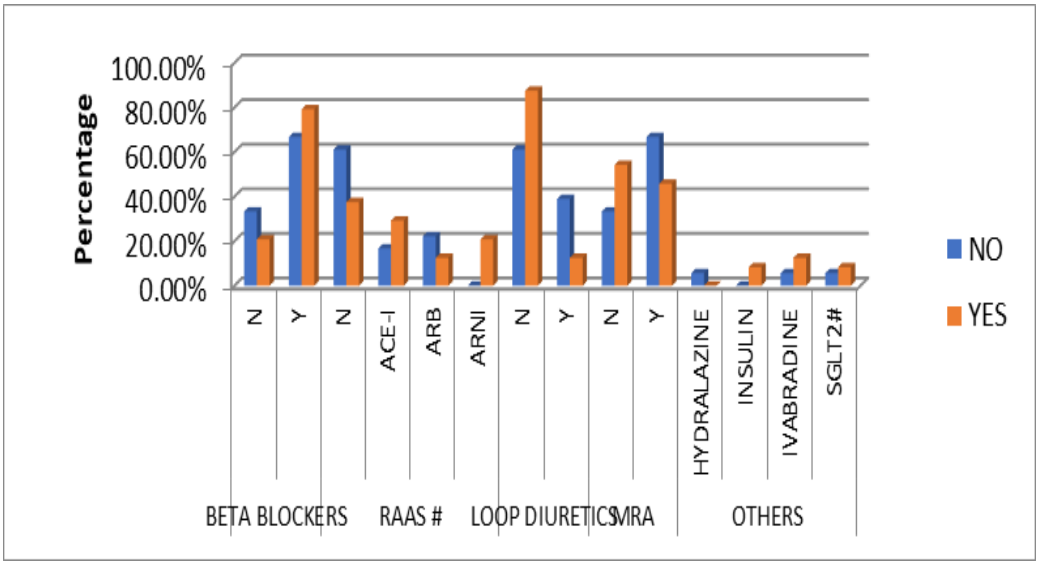

FIGURE 30: TRENDS OF DISCHARGE MEDICATIONS IN RECOVERED/ UNRECOVERED PATIENTS

TABLE 36: COMPARING PARAMETERS IN RECOVERED/ UNRECOVERED PATIENTS

	NO		YES		
	Mean	SD	Mean	SD	Z
HOSPITAL STAY (DAYS)	8.35	2.69	6.29	2.24	2.673
CARPREG II SCORING	5.39	2.43	3.79	1.67	2.528
EF at follow up	38.33	11.63	57.54	3.27	-7.725

This table compares three outcomes—hospital stay, CARPREG II scores, and follow-up EF—between recovered and non-recovered patient groups. Non-recovered patients had a long er mean hospital stay of 8.35 days, compared to 6.29 days in the recovered group and these were statistically significant (p value = 0.011). The difference suggests non-recovered patients required more extended care. Also, the non-recovered group had higher CARPREG II scores, with a mean of 5.39 compared to 3.79 in recovered patients with p value = 0.016(statistically significant). Mean EF at follow-up was 38.33% in non-recovered patients and 57.54% in the recovered group. These findings suggest that recovered patients had lower CARPREG II scores and shorter hospital stays making these metrics potentially valuable for assessing prognosis in this patient population.

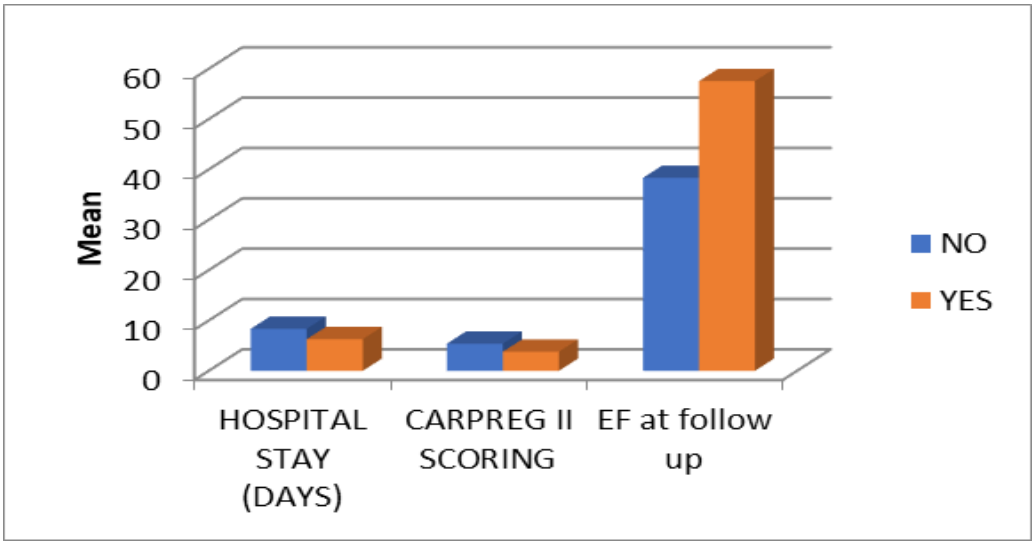

FIGURE 31: COMPARING PARAMETERS IN RECOVERED/ UNRECOVERED PATIENTS

PPCM WITH AND WITHOUT PIH:

Further, Patients diagnosed with PPCM who had associated PIH related disorders or not were compared on basis of their clinical and echocardiographic parameters to evaluate the effect on outcomes of these disorders in patients of PPCM.

Out of 52 patients diagnosed with PPCM, 22 patients had associated PIH (21 had pre-eclampsia and 1 had eclampsia) while remaining 30 patients had no associated PIH disorders. (as mentioned before in table no).

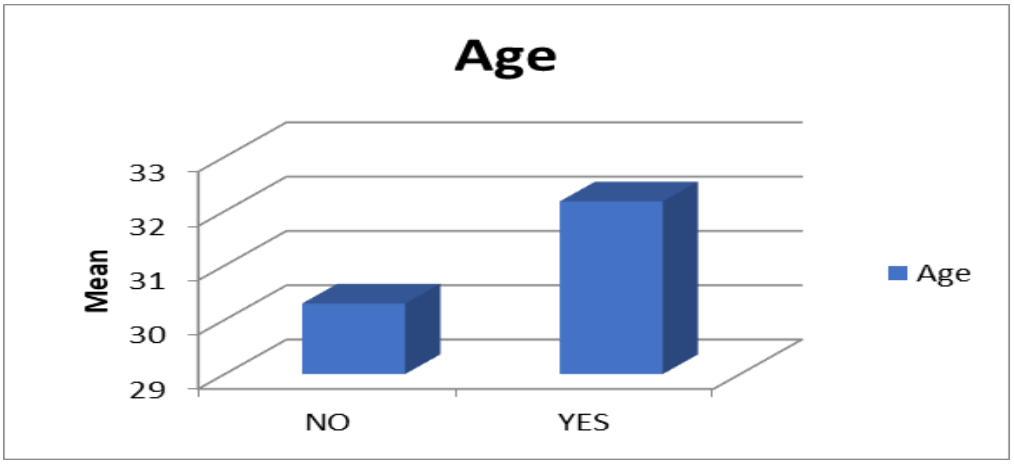

FIGURE 32: DISTRIBUTION OF PPCM WITH AND WITHOUT PIH ACCORDING TO AGE

Mean age was 32.18+-5.56 and 30.30+-4.51 years in patients with and without PIH respectively. Patients who had PIH were slightly older than patients who did not had PIH but this age difference was not found to be statistically significant.

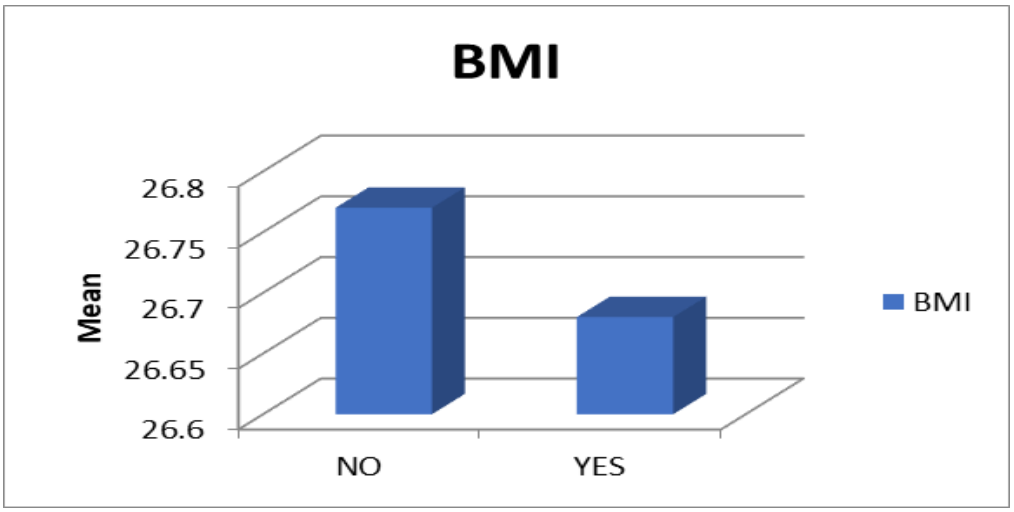

FIGURE 33: DISTRIBUTION OF PPCM WITH AND WITHOUT PIH ACCORDING TO BMI

Mean BMI was comparable in women diagnosed with PPCM both with and without PIH.

TABLE 37: COMAPRING VARIOUS PARAMETERS IN PPCM WITH AND WITHOUT PIH PATIENTS

PIH	NO		YES		Z	p-value
	Mean	SD	Mean	SD		
Age	30.30	4.51	32.18	5.56	-1.346	0.184
BMI	26.77	2.50	26.68	2.34	0.124	0.902
PREGNANCY DURATION (TERMINATION)	33.58	5.47	34.63	3.00	-0.815	0.419
HEMOGLOBIN	10.58	2.35	11.57	1.79	-1.645	0.106
BNP	1091.83	886.69	1273.77	1269.14	-0.609	0.545
EF (%)	29.57	12.00	33.27	8.08	-1.254	0.216
PASP	35.47	12.53	33.05	13.09	0.676	0.502
LVEDD	55.13	5.91	52.86	5.47	1.411	0.164
LVESD	39.27	6.29	36.41	6.65	1.580	0.120
HOSPITAL STAY (DAYS)	6.37	3.91	6.48	2.77	-0.110	0.913
CARPREG II SCORING	4.97	2.27	4.55	2.02	0.693	0.491

Patients who had PIH had higher baseline ejection fraction 33.27+-8.08% as compared to PPCM patients without PIH (29.57+-12.00%). Though this numerical trend infers that patients with PPCM and PIH have better baseline EF's but this was not statistically significant finding. (p value 0.216)

PPCM Patients with PIH had smaller baseline LVEDD as compared to patients without PIH (52.86 vs 55.13 mm) but this was also not a significant finding. (p value 0.164)

Mean CARPREG II score was comparable in both the group of these patients.

The table above shows that none of the variables were statistically significant among patients of PPCM who had PIH or not.

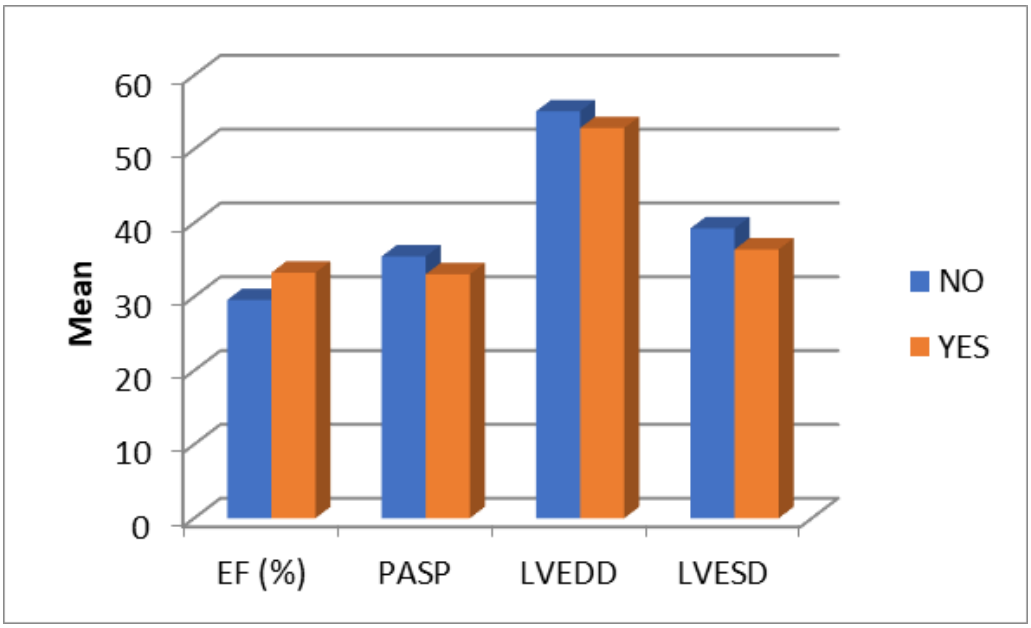

FIGURE 34: COMAPRING VARIOUS PARAMETERS IN PPCM WITH AND WITHOUT PIH PATIENTS

TABLE 38: MATERNO-FETAL OUTCOMES IN PPCM WITH AND WITHOUT PIH PATIENTS

		PIH				Total	Chi-square value	p-value
		N		Y				
FGR	N	29	96.7%	20	90.9%	49	0.774	0.379
	Y	1	3.3%	2	9.1%	3		
PPH	N	26	86.7%	20	90.9%	46	0.224	0.636
	Y	4	13.3%	2	9.1%	6		
PRE-TERM	N	17	56.7%	4	18.2%	21	7.808	0.009
	Y	13	43.3%	18	81.8%	31		
PPROM	N	30	100.0%	21	95.5%	51	1.39	0.423
	Y	0	0.0%	1	4.5%	1		
FETAL/PERINATAL COMPLICATIONS	IUD	5	15.15%	5	20.0%	10	0.237	0.888
	LOW BIRTH WEIGHT	10	30.3%	7	28.0%	17		
	NONE	18	54.5%	13	52.0%	31		
PRIMARY OUTCOMES	BIVENTRICULAR FAILURE	0	0.0%	1	4.5%	1	3.018	0.389
	LEFT VENTRICULAR FAILURE	23	77.7%	18	81.8%	41		
	MATERNAL DEATH	7	23.3%	2	9.1%	9		
	NONE	0	0.0%	1	4.5%	1		
MECHANICAL VENTILATOR	N	23	76.7%	20	90.9%	43	1.799	0.272
	Y	7	23.3%	2	9.1%	9		
MORATILTY	N	22	73.3%	20	90.9%	42	2.524	0.161
	Y	8	26.7%	2	9.1%	10		

It was found that among obstetric complications, preterm birth was significantly more common in PPCM patients with PIH (81.8%) as compared to PPCM patients without PIH (43.3%). This was a significant finding with p value 0.009.

FGR was common in PIH group of PPCM patients as compared to patients without PIH (9.1% vs 3.3%) but this was only a numerical trend and results were not statistically significant.

Similarly, mortality amongst the groups was higher in patients without PIH (26.7%) as compared to PPCM patients without PIH (9.1%) but this finding was not statistically significant.

TABLE 39: LV RECOVERY IN PPCM WITH AND WITHOUT PIH PATIENTS

		PHI				Total	Chi-square value	p-value
		N		Y				
RECOVERED	N	10	33.3%	8	36.4%	18	0.127	0.764
	Y	12	40.0%	12	54.5%	24		

LV Recovery was observed to be more in PPCM patients with PIH (54.5%) as compared to PPCM without PIH (40%). This was only a numerical trend and p value was not significant for these results.

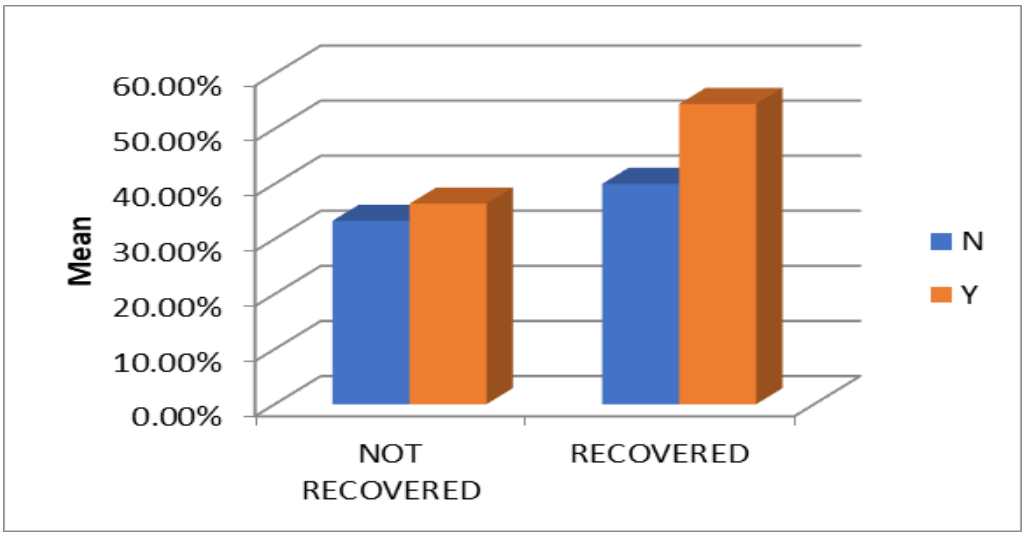

FIGURE 35: LV RECOVERY IN PPCM WITH AND WITHOUT PIH PATIENTS

HYPERTENSIVE DISORDERS OF PREGNANCY(HDP): This group of patients included those with known history of hypertension prior to current pregnancy or PIH patients who were diagnosed to have high blood pressure after 20 weeks of gestation without evidence of LV dysfunction.

TABLE 40: DISTRIBUTION OF PATIENTS ACCORDING TO AGE GROUP

		HDP	
		No. of cases	%age
AGE GROUP	< 25	3	6.7%
	26-30	10	22.2%
	31-35	16	35.6%
	36-40	8	17.8%
	> 40	8	17.8%
Total		45	100.0%

Mean age and gestational period of patients diagnosed with HDP was 34.33+-6.24 years and 33.47+-4.66 weeks respectively.

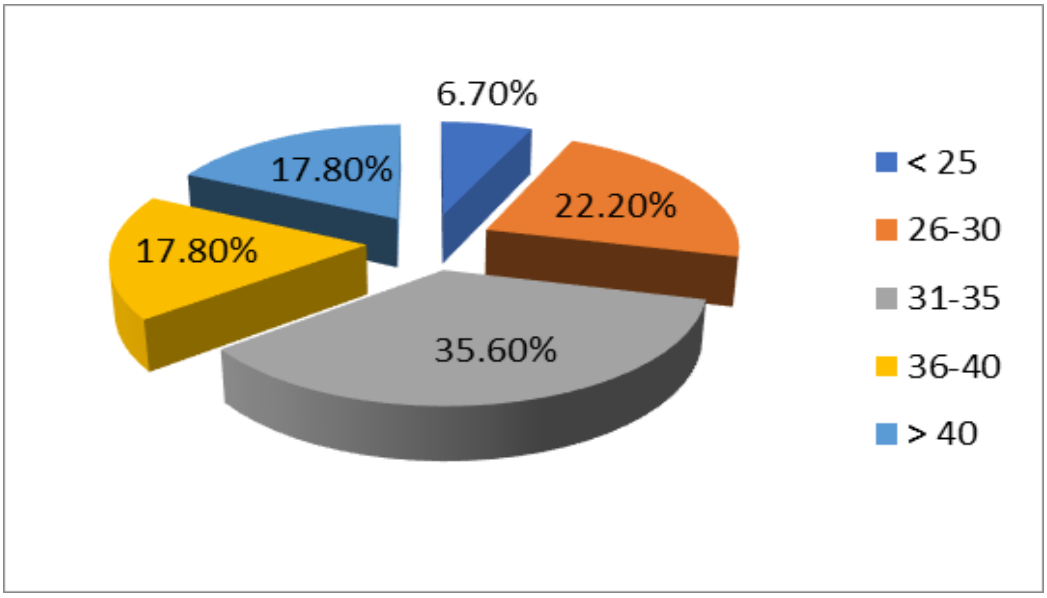

FIGURE 36: DISTRIBUTION OF PATIENTS ACCORDING TO AGE GROUP

TABLE 41: DISTRIBUTION OF HDP PATIENTS ACCORDING TO PARITY

		HDP	
		No. of cases	%age
Parity	MULTI	11	24.4%
	PRIMI	34	75.6%
Total		45	100.0%

Among patients with hypertensive disorders in pregnancy (HDP), 75.6% were primigravida (first-time mothers), and rest 24.4% were multigravida (those with prior pregnancies). As known in literature, primiparity is a known risk factor for HDP.

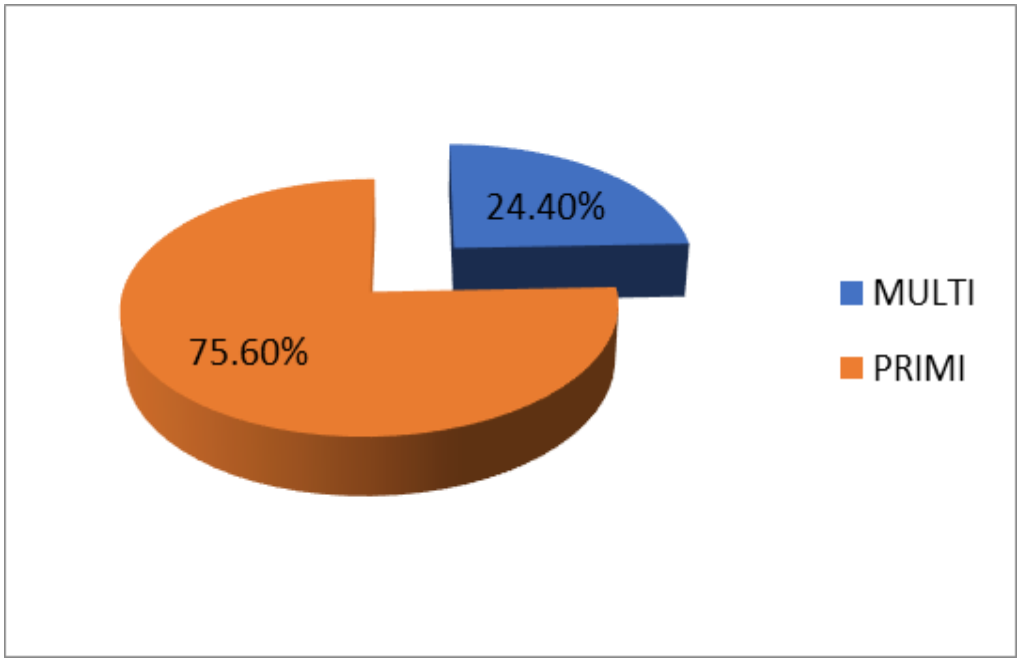

FIGURE 37: DISTRIBUTION OF HDP PATIENTS ACCORDING TO PARITY

TABLE 42: DISTRIBUTION OF HDP PATIENTS ACCORDING TO BMI

		HDP	
		No. of cases	%age
BMI	< 22.9	1	2.2%
	23-24.9	7	15.6%
	25-29.9	29	64.4%
	> 30	8	17.8%
Total		45	100.0%

82.2% of patients diagnosed with HDP had BMI>25kg/m2 which infers that obesity is a common finding in these patients.

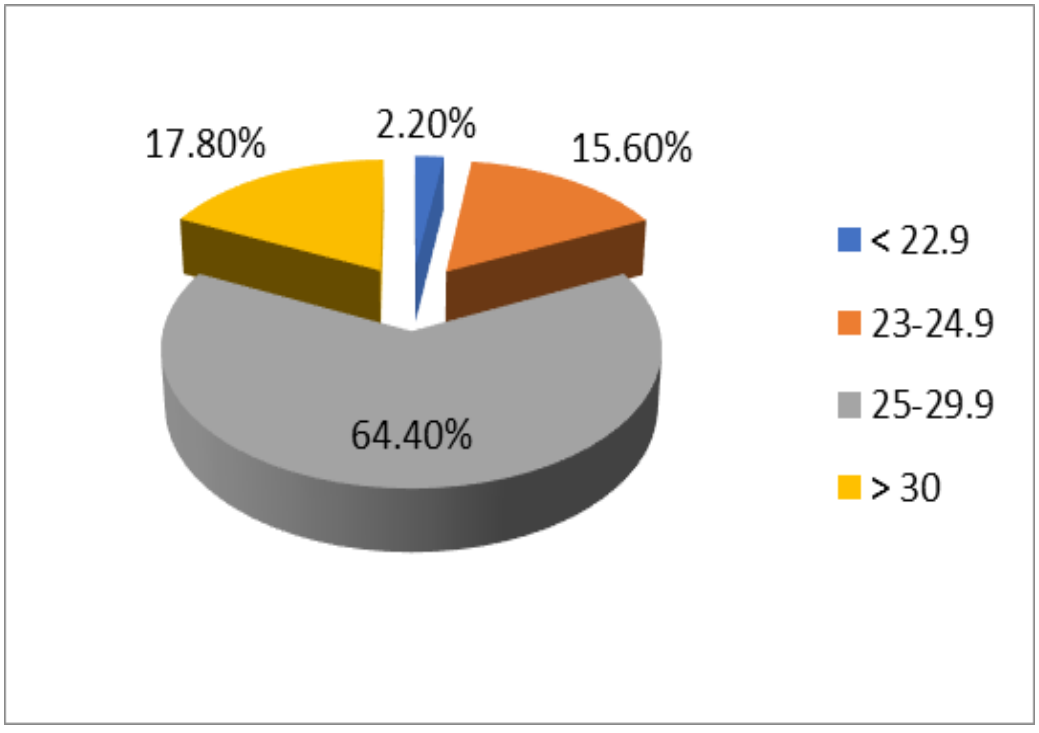

FIGURE 38: DISTRIBUTION OF HDP PATIENTS ACCORDING TO BMI

Out of 45 patients; 7 patients had twin pregnancies among patients who had HDP; 12 patients had history of abortions in the past who had HDP; and 7 patients had history of IVF treatment in current pregnancy. 82.2% patients delivered via LSCS and rest via vaginal delivery.

TABLE 43: VARIOUS COMORBIDITIES IN HDP PATIENTS

		HDP	
		No. of cases	%age
HYPERTENSION	N	27	60.0%
	Y	18	40.0%
HYPOTHYROIDISM	N	40	88.9%
	Y	5	11.1%
DM	N	42	93.3%
	Y	3	6.7%

This table provides the distribution of various comorbidities among patients diagnosed with Hypertensive Disorders of Pregnancy (HDP) and it showed that chronic Hypertension (who were known cases of hypertension antepartum or diagnosed before 20 weeks of gestation) is present in 40.0% of patients with HDP.

TABLE 44: DISTRIBUTION OF PIH PATIENTS

	DIAGNOSIS	HDP	
		No. of cases	%age
GESTATIONAL HTN	N	39	86.7%
	Y	6	13.3%
PRE-ECLAMPSIA	N	33	73.3%
	Y	12	26.7%
PRE-ECLAMPSIA WITH SEVERE FEATURES	N	24	53.3%
	Y	21	46.7%
ECLAMPSIA	N	44	97.8%
	Y	1	2.2%

This table shows the distribution of PIH patients including gestational hypertension, pre-eclampsia (with and without severe features), and eclampsia. Pre-eclampsia affects 73.3% of HDP patients, with nearly two-thirds among these cases showing severe features (63.3%). Gestational hypertension was present in 13.3% of HDP cases. Eclampsia is rare and seen in only 2.2% of HDP patients.

4 patients had co-existing GDM among these 45 patients.

Additionally, 16 patients had chronic hypertension with superimposed PIH.

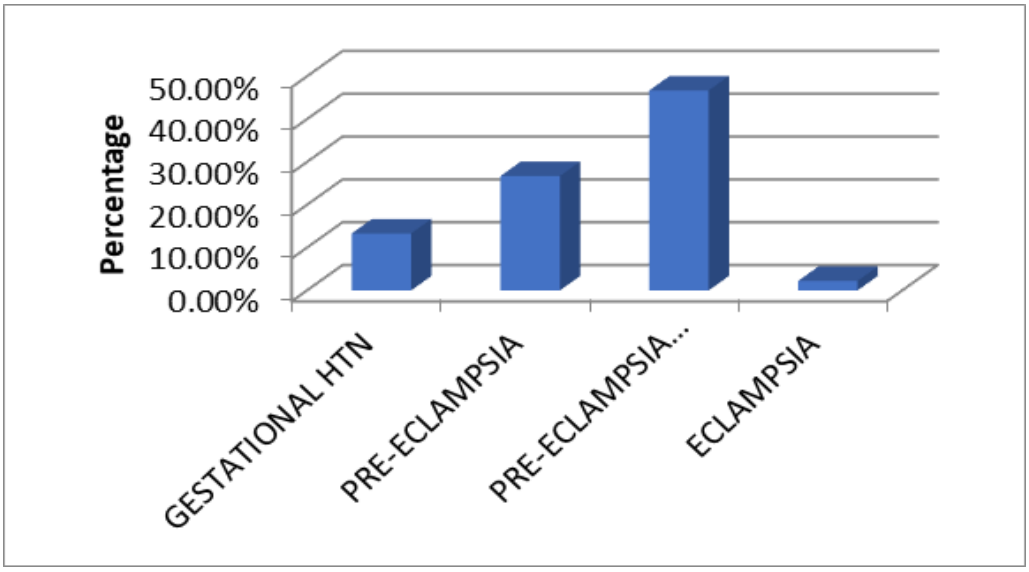

FIGURE 39: DISTRIBUTION OF PIH PATIENTS

TABLE 45: DIASTOLIC DYSFUNCTION IN HDP PATIENTS

	DIAGNOSIS	HDP	
		No. of cases	%age
DIASTOLIC DYSFUNCTION	NO	28	62.2%
	YES	17	37.8%
	TYPE 1	4	8.9%
	TYPE 2	12	26.7%
	TYPE 3	1	2.2%
Total		45	100.0%

The table above presents the prevalence and types of diastolic dysfunction among patients diagnosed with Hypertensive Disorders of Pregnancy (HDP) and it showed that Diastolic dysfunction was present in 37.8% of these patients, indicating that nearly 1 in 3 patients with HDP experience some form of diastolic dysfunction. Among those with diastolic dysfunction, Type 2 is the most prevalent, affecting 26.7% of total HDP patients. These findings suggest that diastolic dysfunction, particularly in its moderate form (Type 2), is a frequent cardiovascular complication in HDP patients, which may have implications for monitoring and managing cardiac function in this population.

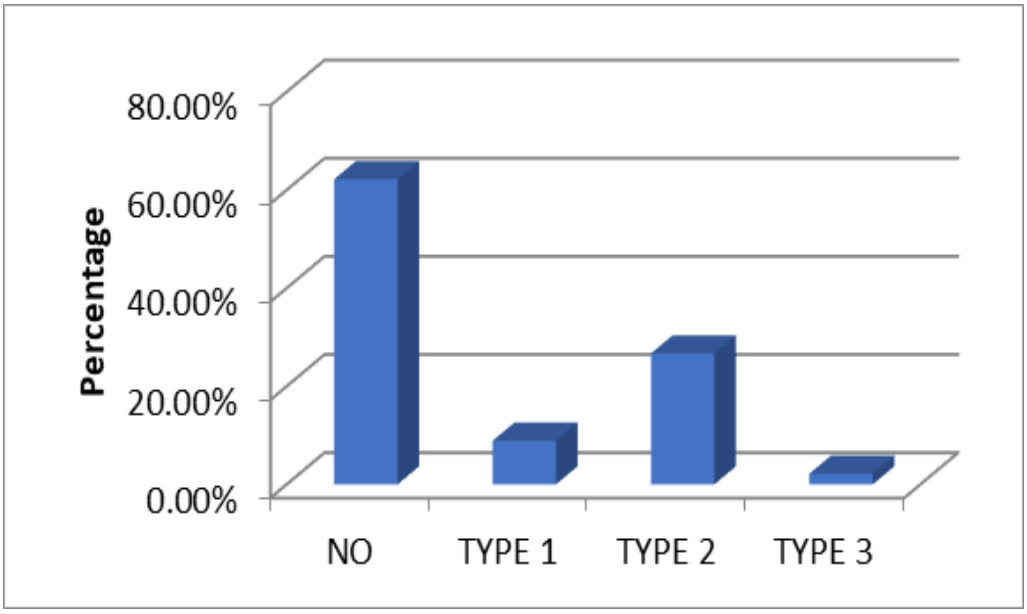

FIGURE 40: DIASTOLIC DYSFUNCTION IN HDP PATIENTS

MATERNAL OUTCOMES:

1 patient died on post-partum (Day 2) who was delivered outside our hospital via LSCS and was referred to our tertiary centre in a very sick state due to severe PPH. Remaining 44 patients were discharged in satisfactory condition on antihypertensives.

TABLE 46: OBSTETRIC OUTCOMES IN HDP PATIENTS

		HDP	
		No. of cases	%age
FGR	N	34	75.6%
	Y	11	24.4%
MACROSMIA	N	44	97.8%
	Y	1	2.2%
OLIGOHYDROAMNIOS	N	42	93.3%
	Y	3	6.7%
POLYHYDRAMNIOS	N	44	97.8%
	Y	1	2.2%
PPH	N	42	93.3%
	Y	3	6.7%
PRE-TERM	N	19	42.2%
	Y	26	57.8%

Among 45 patients, Preterm birth was the most frequent complication in 57.8% of HDP pregnancies followed by fetal growth restriction (FGR) (24.4%) Oligohydramnios and postpartum hemorrhage (PPH) were present in 6.7% of cases each. Macrosomia and polyhydramnios were less common, each occurring in just 2.2% of cases.

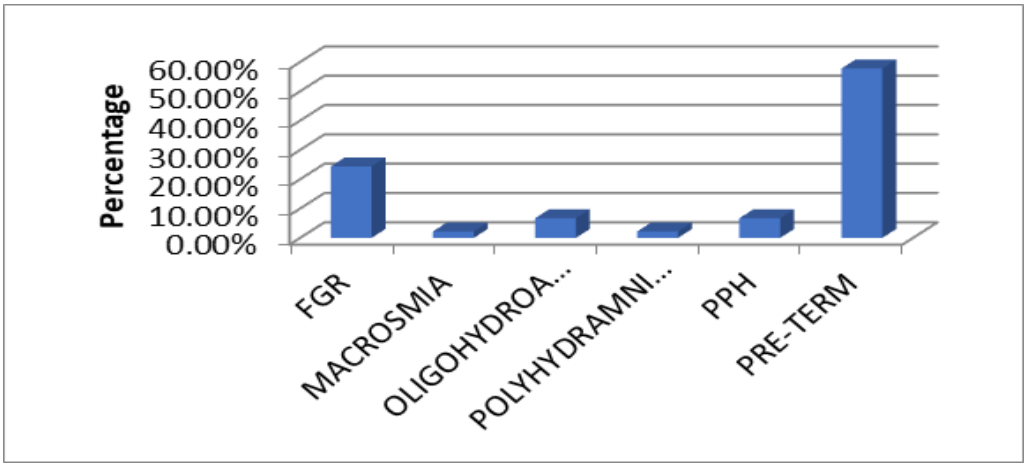

FIGURE 41: OBSTETRIC OUTCOMES IN HDP PATIENTS

TABLE 47: FETAL OUTCOMES IN HDP PATIENTS

		HDP	
		No. of cases	%age
FETAL/PERINATAL COMPLICATIONS	IUD	5	9.6%
	LOW BIRTH WEIGHT	25	48.1%
	MISSED ABORTION	1	1.9%
	NONE	21	40.4%
Total		52	100.0%

Out of 52 total pregnancies in these patients (including twin pregnancies in 7 patients), the fetal and perinatal outcomes exhibited substantial variability. A notable issue was low birth weight, which was observed in 48.1% of cases, and intrauterine demise (IUD) occurred in 9.6% of cases. The abortion of one patient was overlooked at 12 weeks of gestation.

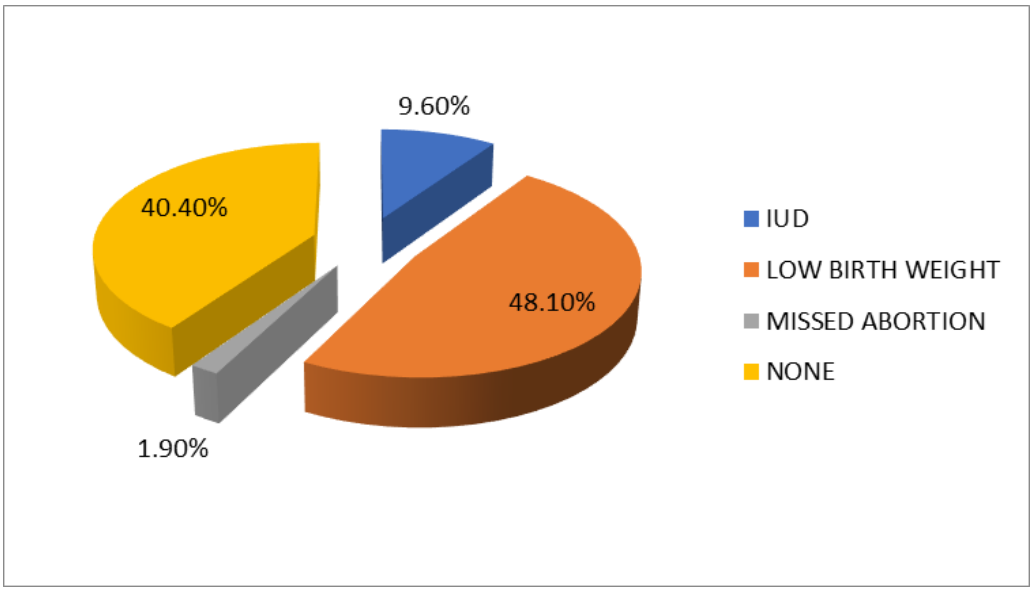

FIGURE 42: FETAL OUTCOMES IN HDP PATIENTS

VALVULAR HEART DISEASE:

All patients who had known valvular heart disease or were operated for VHD in the past along with newly diagnosed cases were enrolled. Out of 16 patients; Fifteen patients had a rheumatic heart disease; with all patients having mitral valve involvement and one patient had both mitral and aortic valve involvement. No patients experienced tricuspid or pulmonary valve involvement due to RHD. Three patients were admitted with a post-mitral valve replacement (MVR) status and all three had metallic valves. One patient had vegetation on the anterior and posterior mitral leaflets, which was associated with embolic complications such as acute cerebrovascular accident (CVA) and pulmonary infarcts.

Mean age of 16 patients was 32.88+-4.515 years. Mean BMI was 23.94+-3.172 kg/m2.

Mean hemoglobin was 10.87+-1.158 gm/dl.

9 patients were known case of RHD before current pregnancy while rest were diagnosed during evaluation in current pregnancy.

TABLE 48: DISTRIBUTION OF VHD PATIENTS ACCORDING TO PARITY

	DIAGNOSIS	VHD	
		No. of cases	%age
Parity	MULTI	7	43.8%
	PRIMI	9	56.3%

3 patients had history of abortions in past and none had twin pregnancy.

11 patients delivered via LSCS and 5 patients had vaginal delivery.

Mean CARPREG II score of these patients was 2.06+-2.11

ECHOCARDIOGRAPHIC PARAMETERS:

All 3 Post MVR showed normal functioning prosthetic metallic valve with normal LVEF.

6 patients had isolated mitral stenosis which came out to be most common lesion in these patients. 2 patients had isolated mitral regurgitation. 3 patients had mixed mitral valve disease. 1 patient had mixed mitral valve disease along with aortic regurgitation 1 patient had endocarditis of mitral valve. No previous risk factors were found in this patient.

TABLE 49: MATERNAL, OBSTETRIC AND FETAL OUTCOMES IN PREGNANT RHD PATIENTS

	DIAGNOSIS	VHD	
		No. of cases	%age
AKI	N	15	93.8%
	Y	1	6.3%
OTHERS	EMBOLIC STROKE	1	6.3%
ANHYDROAMNIOS	N	15	93.8%
	Y	1	6.3%
OLIGOHYDROAMNIOS	N	15	93.8%
	Y	1	6.3%
PRE-TERM	N	9	56.3%
	Y	7	43.8%
FETAL/PERINATAL COMPLICATIONS	IUD	0	0.0%
	LOW BIRTH WEIGHT	3	18.8%
	MISSED ABORTION	1	6.3%

This table presents outcomes for patients with valvular heart disease (VHD), and it showed that Preterm deliveries were seen in 43.8% of patients, indicating a notable incidence of early deliveries, Low birth weight was seen in 18.8% of cases and 1 patient had AKI, embolic stroke, an-hydroamnios, oligo-hydroamnios and missed abortion each. This data suggests that preterm birth and low birth weight are more frequent among VHD patients, potentially due to maternal cardiovascular strain impacting fetal outcomes.

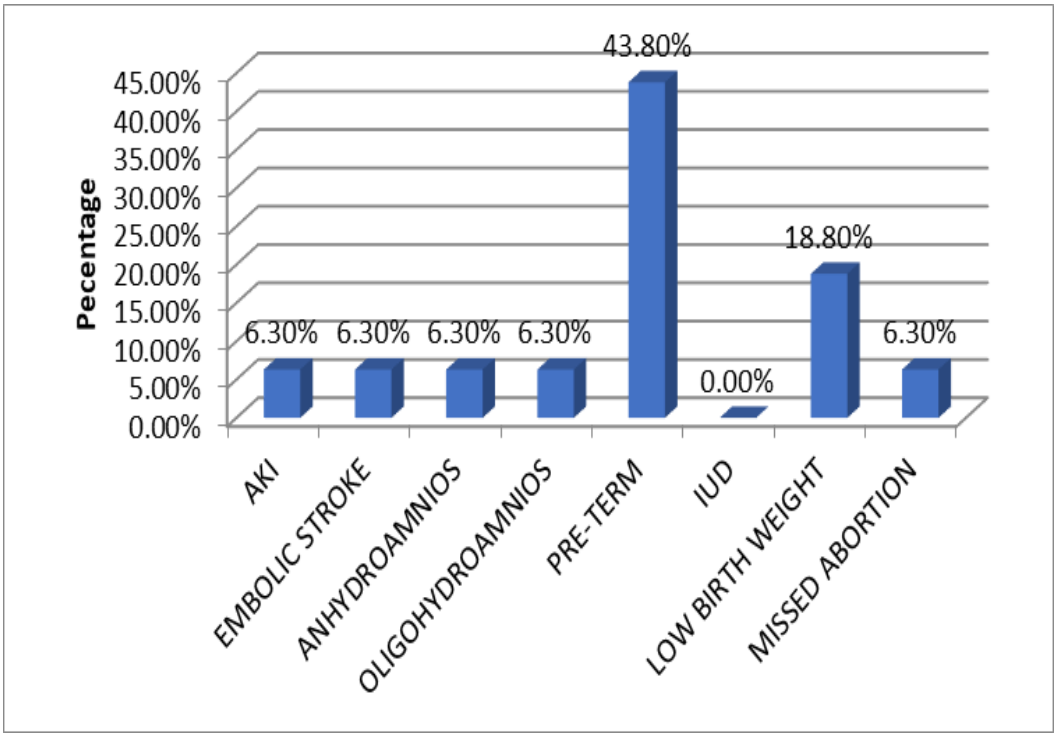

FIGURE 43: MATERNAL, OBSTETRIC AND FETAL OUTCOMES IN PREGNANT RHD PATIENTS

THEIR WAS NO MORTALITY IN THESE PATIENTS.

CONGENITAL HEART DISEASE:

We enrolled all patients with known congenital heart disease, those who had undergone previous CHD operations, and newly diagnosed cases. Out of the 9 patients, 5 had shunt lesions, consisting of 2 operated cases of VSD closure, 1 unrepaired VSD, 1 PDA, and 1 PFO. These were followed by 2 patients with cyanotic heart disease-TOF physiology, consisting of 1 repaired and 1 unrepaired case. The remaining 2 patients had ebstein anamoly and a biscuspid aortic valve.

Mean age of 9 patients was 29.89+-3.52 years. Mean BMI was 25.0+-1.87 kg/m2.

Mean hemoglobin was 10.92+-1.27 gm/dl.

8 patients were known case of CHD before current pregnancy while remaining 1 was diagnosed to have BAV with severe aortic stenosis during evaluation in current pregnancy.

All 9 patients who had CHD were primiparous and none were multiparous.

No patient had history of abortions in past or had twin pregnancy.

5 patients delivered via LSCS and 4 patients had vaginal delivery.

MATERNAL, OBSTETRIC AND FETAL OUTCOMES:

Out of 9 patients, 1 each had preterm birth and low birth weight while 4 patients had missed abortion.

THEIR WAS NO MORTALITY IN THESE PATIENTS.

THROMBOTIC DISORDERS:

Mean age of these patients was 32.67+-7.92 years. 2 patients were diagnosed cases of lower limb DVT and remaining 4 had acute DVT in current pregnancy (50% had dreaded complication of pulmonary embolism). Mean BMI of these patients was 28.50 kg/m2 which infers that obesity is a risk factor for occurrence of thrombotic disorders in pregnant patients or general population.

Among various outcomes, out of 6 patients; 3 had preterm birth and 1 patient had IUD.

There was no mortality in these patients but mean hospital stay was highest among all the patients (7.60 days).

MORTALITY DATA:

Out of 135 patients, 11 patients died (10 during index hospitalization and 1 died after discharge) with mortality rate 8.14 %. The baseline characteristics of these patients were studied and correlated with various outcomes to estimate the burden of these factors and to improve the quality of maternal care in these patients. Total maternal deaths recorded over 18 months was 15, out of which 11 were due to cardiac ailments.

In our study, 90.9% of patients who died were in age group from 26-35 years, therefore it can be inferred that mortality cannot be attributed to extreme ages in pregnant women with heart disease.

Further, the proportion of primiparous or multiparous patients was equal in the patients who died and it was not a mortality predictor as per our study.

81.8% patients had BMI >=25 kg/m2 who died as compared to 70.2% patients in the alive group but this was just a numerical trend and not found to be statistically significant.

63.6% of patients who died were diagnosed to have cardiac ailment post-partum as compared to 32.3% in the alive patients and it was a significant finding (p value 0.049). Thus, it was concluded that postpartum diagnosis of heart disease which may be due to latent asymptomatic phase or poor coordination between the cardiologist and the primary team in the peripartum period can be associated with mortality predictor.

TABLE 50: MORTALITY DATA ACCORDING TO PATIENTS CHARACTERICTICS

		MORATILTY				Total	Chi-square value	p-value
		N		Y				
AGE GROUP	< 25	14	11.3%	0	0.0%	14	4.729	0.316
	26-30	36	29.0%	6	54.5%	42		
	31-35	43	34.7%	4	36.4%	47		
	36-40	18	14.5%	1	9.1%	19		
	> 40	13	10.5%	0	0.0%	13		
POST PARTUM (DAYS) GP	N	84	67.7%	4	36.4%	88	4.384	0.049
	Y	40	32.3%	7	63.6%	47		
Parity	MULTI	39	31.5%	6	54.5%	45	2.425	0.179
	PRIMI	85	68.5%	5	45.5%	90		
ABOORTION	N	98	79.0%	9	81.8%	107	0.048	0.827
	Y	26	21.0%	2	18.2%	28		
BMI	1	11	8.9%	0	0.0%	11	1.381	0.71
	2	26	21.0%	2	18.2%	28		
	3	73	58.9%	8	72.7%	81		
	4	14	11.3%	1	9.1%	15		
mode of delievery	LSCS	95	76.6%	6	54.5%	101	4.634	0.060
	VAGINAL DELIVERY	21	16.9%	5	45.5%	26		
	VAGINAL EXPULSION AND CHECK CURETTAGE	8	6.5%	0	0.0%	8		
Total		124	100.0%	11	100.0%	135		

TABLE 51: MORTALITY DATA ACCORDING TO CLINICAL PRESENTATION

		MORATILTY				Total	Chi-square value	p-value
		N		Y				
SHORTNESS OF BREATH	N	71	57.3%	1	9.1%	72	9.418	0.002
	Y	53	42.7%	10	90.9%	63		
PALPITATIONS	N	117	94.4%	11	100.0%	128	0.655	0.418
	Y	7	5.6%	0	0.0%	7		
GTCS	N	122	98.4%	11	100.0%	133	0.180	0.671
	Y	2	1.6%	0	0.0%	2		
FEVER	N	119	96.0%	10	90.9%	129	0.609	0.435
	Y	5	4.0%	1	9.1%	6		
POST PARTUM HEMORRHAGE	N	121	97.6%	9	81.8%	130	7.039	0.053
	Y	3	2.4%	2	18.2%	5		
INCREASED SUGARS	N	121	97.6%	10	90.9%	131	1.564	0.291
	Y	3	2.4%	1	9.1%	4		
SHOCK	N	120	96.8%	7	63.6%	127	19.902	0.001
	Y	4	3.2%	4	36.4%	8		
OTHERS	BICUSPID AV- ASYMPTOMATIC	1	0.8%	0	0.0%	1	0.655	0.995
	CHEST PAIN	2	1.6%	0	0.0%	2		
	PRESYNCOPE	1	0.8%	0	0.0%	1		
	RIGHT HEMIPARESIS	1	0.8%	0	0.0%	1		
NYHA CLASS(ADMSN)	I	7	5.6%	0	0.0%	7	35.57	0.001
	II	24	19.4%	0	0.0%	24		
	III	21	16.9%	2	18.2%	23		
	IV	11	8.9%	8	72.7%	19		
	NA	61	49.2%	1	9.1%	62		

90.9% of patients who died had dyspnea at admission as compared to 42.7% in alive patients and this was statistically significant. Taking NYHA class into consideration, 72.7% of died patients had class IV symptoms as compared to 8.9% alive patients and this difference was found to be significant. 36.4% of patients who died presented in shock at admission as compared to 3.2% in alive patients which was also a significant finding. Thus, it was concluded that few symptoms at presentation like presence of severe dyspnoea and shock can be corelated as mortality predictors in pregnant patients with heart diseases.

TABLE 52: SHOWING OTHER COMORBIDITIES AND ASSOCIATIONS

		MORATILTY				Total	Chi-square value	p-value
		N		Y				
HYPERTENSION	N	94	75.8%	9	81.8%	103	0.202	0.653
	Y	30	24.2%	2	18.2%	32		
BRONCHIAL ASTHMA	N	123	99.2%	11	100.0%	134	0.089	0.765
	Y	1	0.8%	0	0.0%	1		
HYPOTHYROIDISM	N	109	87.9%	11	100.0%	120	1.497	0.611
	Y	15	12.1%	0	0.0%	15		
HYPERTHYROIDISM	N	124	100.0%	10	90.9%	134	11.357	0.081
	Y	0	0.0%	1	9.1%	1		
DM	N	119	96.0%	10	90.9%	129	0.609	0.435
	Y	5	4.0%	1	9.1%	6		
GDMA2	N	116	93.5%	11	100.0%	127	0.754	0.385
	Y	8	6.5%	0	0.0%	8		
GESTATIONAL HTN	N	117	94.4%	11	100.0%	128	0.655	0.418
	Y	7	5.6%	0	0.0%	7		
PRE-ECLAMPSIA	N	98	79.0%	11	100.0%	109	2.857	0.122
	Y	26	21.0%	0	0.0%	26		
PRE-ECLAMPSIA WITH SEVERE FEATURES	N	98	79.0%	8	72.7%	106	0.238	0.626
	Y	26	21.0%	3	27.3%	29		

ECLAMPSIA	N	122	98.4%	11	100.0%	133	0.18	0.671
	Y	2	1.6%	0	0.0%	2		
SEPSIS	N	120	96.8%	5	45.5%	125	38.798	0.001
	Y	4	3.2%	6	54.5%	10		
AKI	N	122	98.4%	11	100.0%	133	0.18	0.671
	Y	2	1.6%	0	0.0%	2		
VTE	N	118	95.2%	11	100.0%	129	0.557	0.455
	Y	6	4.8%	0	0.0%	6		
PPH	N	117	94.4%	8	72.7%	125	6.891	0.036
	Y	7	5.6%	3	27.3%	10		
PRE-TERM	N	56	45.2%	8	72.7%	64	3.079	0.115
	Y	68	54.8%	3	27.3%	71		
FETAL/PERINATAL COMPLICATIONS	IUD	12	8.8%	6	54.5%	18	20.484	0.001
	LOW BIRTH WEIGHT	45	32.8%	1	9.1%	46		
	MISSED ABORTION	6	4.4%	0	0.0%	6		
	NONE	74	54.0%	4	36.4%	78		

It was found that 54.5% who died had associated sepsis as compared to only 3.2% in alive group and this was a significant finding (p value 0.001).

Patients who died also had more number of materno-fetal complications like PPH and IUD’s which was also statistically significant.

INVESTIGATIONS:

Mean hemoglobin in patients who died was 9.20 gm/dl as compared to 11.30 in alive patients which was a statistically significant finding, it inferred that anemia is important parameter deciding the fate of these patients. (p value 0.001)

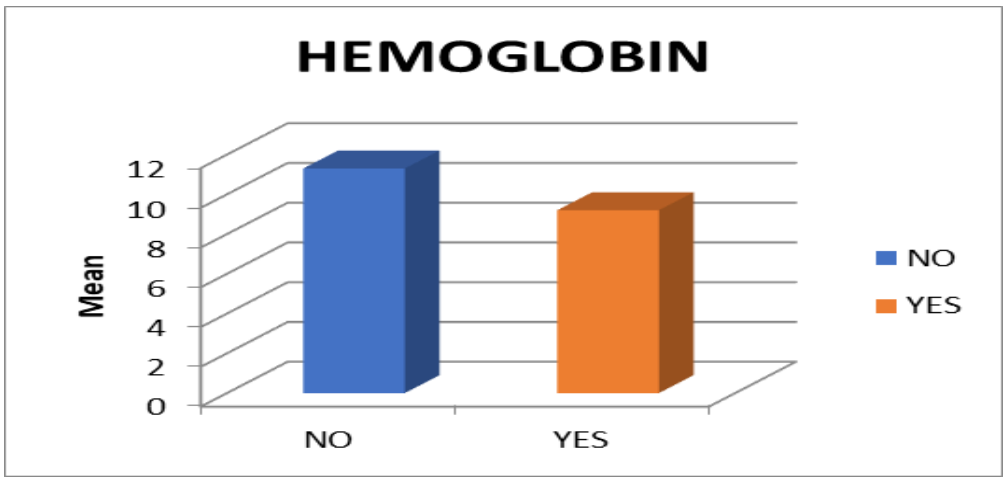

FIGURE 44: SHOWING MEAN HEMOGLOBIN AMONG SURVIVORS AND EXPIRED PATIENTS

10 out of 11 patients who died had PPCM and was associated with moderate to severe left ventricular systolic dyfunction, mean baseline ef in patients who died was 26.91% which was higher than those in alive patients. (p value 0.001)

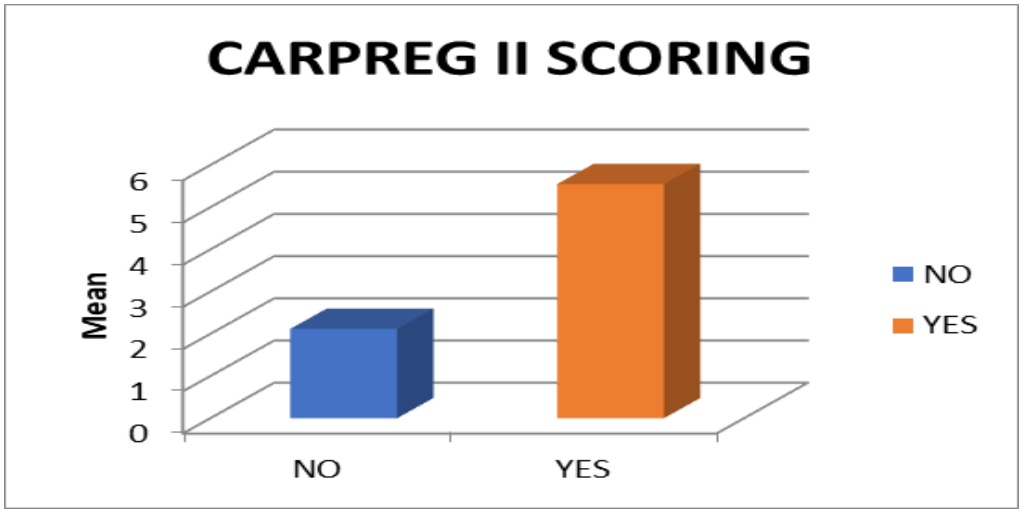

FIGURE 45: SHOWING CARPREG II SCORE OF PATIENTS WHO DIED AND SURVIVED

Also mean CARPREG II score of patients who died was much higher (5.55) as compared to alive patients (2.12). this score can be a strong mortality predictor and needs more studies for corelation. (p value 0.001)

TABLE 53: SHOWING VARIOUS CHARACTERISTICS AMONG SURVIVOR AND DISEASED PATIENTS

MORTALITY	NO		YES		Z	p-value
	Mean	SD	Mean	SD		
QRS	100.01	12.13	108.55	17.55	-2.136	0.035
EF (%)	50.02	14.46	26.91	14.16	5.088	0.001
RVFAC	34.21	2.95	24.91	12.15	6.755	0.001
PASP	30.09	10.74	41.64	11.02	-3.404	0.001
LVEDD	48.29	6.19	54.55	5.96	-3.214	0.002
LVESD	31.74	6.48	40.64	7.42	-4.302	0.001
HEMOGLOBIN	11.30	1.54	9.20	2.75	4.008	0.001
BNP	821.04	977.42	1646.36	950.83	-2.571	0.012
HOSPITAL STAY (DAYS)	6.35	3.40	3.36	4.59	2.674	0.009
CARPREG II SCORING	2.12	2.46	5.55	2.42	-4.427	0.001

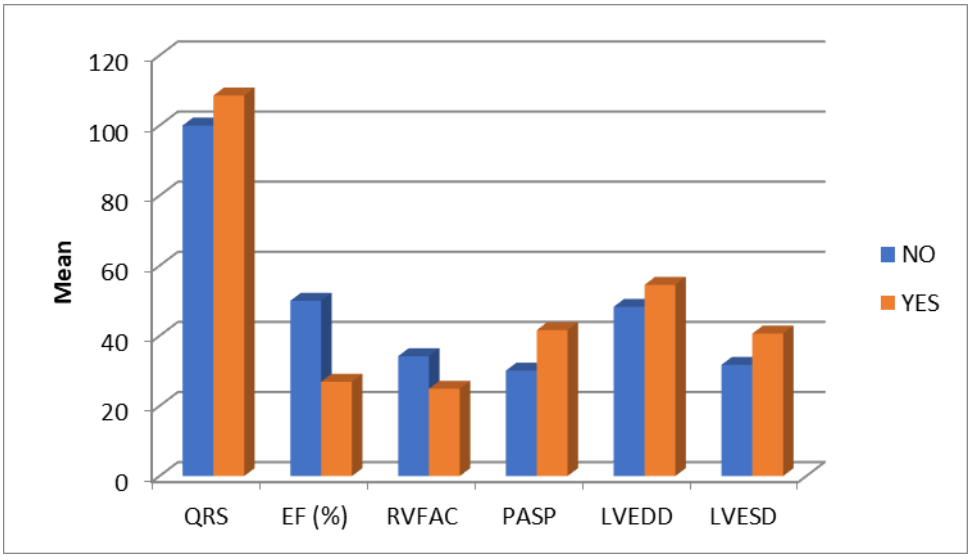

FIGURE 46: SHOWING VARIOUS CHARACTERISTICS AMONG SURVIVOR AND DISEASED PATIENTS

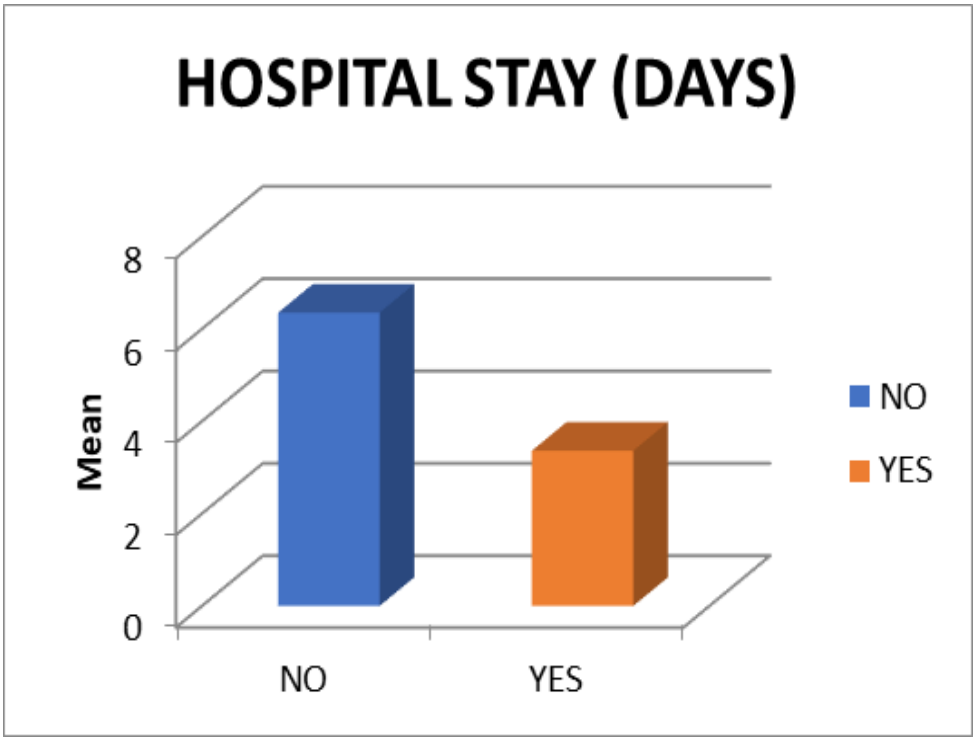

FIGURE 46: SHOWING AVERAGE DURATION OF HOSPITAL STAY FOR DISEASED AND ALIVE PATIENTS

The average hospital stay for deceased patients was 3.36 days, while that of alive patients was 6.35 days. This could be attributed to the fact that these patients were extremely ill at the time of admittance or were referred to our tertiary care center at a late stage, resulting in a limited amount of time to manage them.

DISCUSSION

The study was conducted at Dayanand Medical College and Hospital in Ludhiana, Punjab, which is a tertiary care center in North India. All pregnant patients admitted with various cardiac ailments were included from the cardiology and obstetrics wards, as well as from cardiac and medical ICUs. The baseline characteristics of these patients were recorded, along with electrocardiographic and echocardiographic abnormalities. The patients were followed up for six months, and CARPREG II scores were calculated. These scores were used to predict recovery, maternal mortality, and morbidity, and to assess obstetric and perinatal complications or outcomes.

In total, 135 pregnant patients (study subjects) were enrolled and of these, 52 were diagnosed with peripartum cardiomyopathy which was found to be the most common cardiac ailment in our subjects. It was not in corroboration with a previous meta-analysis done in South Asia by Shrestha et al.[43] This may be because our hospital is a tertiary care center majorly serving very sick patients who are being referred in the late peri partum period. Our study had 16 patients with valvular heart disease, 9 patients with congenital heart disease, 6 patients with thrombotic diseases, 2 patients with idiopathic pulmonary arterial hypertension (PAH), and 1 patient each with hypertrophic cardiomyopathy and DCMP (newly diagnosed) and 3 patients of myocarditis. The prevalence of common cardiac ailments like RHD was less as compared to previous Indian subcontinent studies because our centre is a private tertiary care centre where more patients from the higher socio-economic group have better access while RHD is more common in lower socio-economic strata. Among RHD patients, isolated mitral stenosis was the most common lesion identified. This finding aligns with previous study done by Abhilashi et al (47).

The prevalence of heart disease in pregnant patients with and without HDP was 7.4% and 4.9% respectively.

TABLE 54: THE BELOW TABLE SHOWS THE DISTRIBUTION OF VARIOUS HEART DISEASE IN OUR STUDY (N=90).

DIAGNOSIS	No. of cases	%age
PPCM	52	57.77%
VHD	16	17.77%
CHD	9	10.0%
VTE	6	6.66%
OTHER	7	7.77%
Total	90	100.0%

Additionally, 45 patients were diagnosed with hypertensive disorders of pregnancy.

The mean age of our subjects was found to be 32.24+-5.66 years with the youngest patient presenting at 19 years of age and the eldest patient at 48 years of age, and the mean gestational period for pregnant females was 33.14+-6.28 weeks; the mean CARPREG II score was 2.40+-2.62.

Further, we tried to emphasize on antepartum follow up's of pregnant females that whether they were booked or un-booked which could affect their maternal and fetal outcomes and we found that >2/3rd of all pregnancies were un-booked; despite that patients with VHD and CHD received better cardiac care in antepartum period and these patients showed better trends in terms of less severe clinical presentation and improved materno-fetal outcomes vis-à-vis PPCM and HDP; among these fetal outcomes were significant statistically (p value 0.001).

We found that VHD and CHD patients had lower BMI as compared with PPCM and HDP (p value 0.004).

We found that VHD/CHD/VTE patients had significantly a greater number of vaginal expulsion and curettage as compared to PPCM and HDP patients due to higher number of medically contraindicated pregnancies in these patients (p value 0.003).

Diastolic dysfunction was significantly less in number in VHD/CHD/VTE patients (p value 0.005).

We found a significant association of mortality among PPCM and HDP vis-à-vis CHD/VHD/VTE patients; it was inferred that later subjects had lower mortality rate.

PERIPARTUM CARDIOMYOPATHY:

We found that a significant number of these patients were diagnosed in the postnatal period (56.4%)- mostly in 1st month of post-partum period which contrasts with the findings of another study done by Shakya et al [32] that showed a much higher number (91%) of patients diagnosed in the post-partum period. Primiparous women were more prevalent in our subjects which contrasts with the results of previous studies which confirmed multiparity as risk factor for PPCM. Most patients (71.2%) were in NYHA class III/IV at time of admission. 76.9% of patients were delivered via LSCS, and the remaining via vaginal delivery. The proportion of cesarean deliveries was higher in all pregnancies delivered at our hospital.

RECOVERY PREDICTORS OF PPCM:

Recovery was seen in 57.14% of our subjects. This was almost consistent with a previous study done by Chauhan et al (63.4%) (22). The findings of our study highlighted various predictors between recovered and non-recovered patients of Peripartum Cardiomyopathy (PPCM), specifically emphasizing the role of NYHA functional class at admission. Our study found that NYHA class IV at admission was associated with a significantly higher risk of non-recovery in PPCM patients. Patients admitted with NYHA Class IV had a much higher likelihood of non-recovery (44.4%), compared to only (4.2%) of recovered patients in this class. In contrast, patients in NYHA Class I-III demonstrated a higher likelihood of recovery. These findings were in concordance with other studies like Sliwa et al.[48] and Munir et al.[49] where severe heart failure symptoms have been consistently linked to poorer outcomes in PPCM patients. These results further contribute to the growing understanding of PPCM, particularly in the context of South Asia, where the condition often presents unique clinical challenges due to socioeconomic factors, healthcare access, and genetic predispositions.

In our study, non-recovered patients had a significantly lower mean EF (27.94%) compared to recovered patients (36.67%). The relationship between ejection fraction (EF) and recovery in PPCM has also been noted in previous South Asian studies such as Thakur et al [50] and Hossain et al.[51]

Another significant recovery predictor in our study was the difference in BNP (B-type Natriuretic Peptide) levels among recovered and non-recovered patients. Non-recovered patients had substantially higher BNP levels (1623.89 pg/mL) than recovered patients (584.92 pg/mL), indicating a strong association between elevated BNP and poor recovery. This biomarker, a well-established indicator of cardiac stress, has similarly been highlighted as a prognostic tool in other studies like Imran et al.[52]

We also found that among the patients who had complete recovery at follow up were more likely to have smaller LVEDD (51.42 vs 57.11 mm) and higher gestational period (35.98 vs 33.88 weeks); both the p values were statistically significant. Compared to the studies from Western countries, the findings of PPCM studies in South Asia often emphasize the late presentation of patients and the limited access to advanced healthcare as significant factors influencing outcomes. Additionally, cultural factors, such as delayed reporting of symptoms due to societal expectations around pregnancy and motherhood, may also play a role in poorer outcomes compared to Western cohorts. Identifying high-risk patients based on NYHA class, EF, and BNP levels may improve outcomes, especially when combined with tailored interventions like prompt heart failure therapy and close postpartum monitoring.

OUTCOMES OF PPCM WITH AND WITHOUT PIH:

We found that Preterm birth was the more common in PPCM patients with PIH patients than without (81.8% vs 43.3%); p value 0.009. Riyami et al did a similar study in Oman and found the preterm birth rate was 35.7%.[54] Another Study done by sharma et al [55] showed similar high rates of pre-term birth in these patients.

MATERNAL MORBIDITY AND MORTALITY:

Our study reported high maternal deaths due to associated cardiac ailments. Out of 15 maternal deaths recorded over 18 months, 11 were directly or indirectly related to cardiovascular involvement (73.3%). Previous studies done by Kotit et al[56] showed that cardiovascular disease was the single largest cause of indirect maternal mortality accounting for over 33% of pregnancy-related maternal deaths. Another study done by Khade et al [57] also found a high maternal death rate. Maternal mortality in our subjects was high as compared to previous south Asian studies which might be due to the high referral rate from rural areas and admission of very sick un-booked pregnancies around the peripartum period.

The high mortality rate observed, 8.14%(n=135) aligns with studies from regions where access to early diagnosis and comprehensive maternal care may be limited. Left ventricular failure was the most common (78.8%) primary outcome among patients with Peripartum Cardiomyopathy (PPCM). This is indicative of the substantial influence of PPCM on cardiac function, particularly the left ventricle, which is essential for systemic circulation and is significantly influenced by the heightened physiological demands of pregnancy. It is notable that certain patient demographics and clinical presentations significantly impacted mortality outcomes in cases of PPCM and another maternal cardiac disease. We found that most deceased patients of PPCM were in the 26–35 age range, contradicting the notion that younger or older maternal age independently increases mortality risk in PPCM. We observed that severe dyspnea and shock at presentation were significant predictors of mortality, with 90.9% and 36.4% of deceased patients experiencing severe dyspnea and shock respectively. This is consistent with findings from the IPAC study, [39] which reported that patients in advanced NYHA classes (III or IV) had higher mortality rates in PPCM. The role of NYHA classes as a predictor of outcomes is reinforced by multiple studies, emphasizing that patients with higher NYHA classes at admission may require more intensive monitoring and early intervention. Our study also found a significant association

between postpartum diagnosis and mortality, with 63.6% of deceased patients being diagnosed after delivery (p value 0.049). These findings stress the importance of early and coordinated management between obstetrics and cardiology team to identify cardiac risk factors in the peripartum period.

We found that Anemia had a significant association with mortality (mean hemoglobin 9.2 vs 11.3gm/dl in deceased and alive patients), suggesting that low hemoglobin levels may contribute to poor outcomes. A systemic review done by Cherubin et al [58] found that the prevalence estimates of anemia among patients with PPCM range from 16% to over 40%, and the associated odds of PPCM in pregnant women with anemia are up to five folds higher than among women with normal hemoglobin concentration. Another study done by Ehtesham et al [59] also reported increased mortality in PPCM patients with anemia as compared to patients without anemia.

Sepsis and PIH were significantly associated with mortality, affecting 54.5% and 27.3% of the deceased respectively. This aligns with findings done by Nimbalkar et al [60] in central India, where sepsis was a frequent and severe complication in PPCM.

SUMMARY AND CONCLUSION

- N=135 pregnant females presented with cardiac ailments and HDP; of these peripartum cardiomyopathy was the commonest (38.5%) followed by RHD (11.8%) and CHD (6.66%). This may not be representative of general population given this was a single centred study.
- 74.6% of subjects delivered via caesarean section (like non-cardiac patients). The high prevalence of LSCS in our institute is representative of sicker patients referred to the tertiary care centre for delivery.
- Elderly patients more commonly presented with HDP (mean age 34.33 years) vis-à-vis PPCM (31.10 years) and CHD/VHD/VTE patients (31.97 years) (p value 0.015)
- Amongst the subjects, 2/3rd of all pregnancies were un-booked. It was found that patients with VHD, CHD and VTE were followed up in cardiology OPD's in antepartum period.
- VHD & CHD patients had lower BMI vis-à-vis PPCM and HDP (p value 0.004). On comparing materno-fetal outcomes amongst the booked and un-booked cases especially PPCM and HDP vis-à-vis VHD/CHD/VTE; it was found that there was statistically significant amelioration in fetal outcomes like IUD, FGR and low birth weight of booked patients. (p value 0.001)
- The most common presentation in PPCM patients was dyspnea with 71.2% having NYHA class III/IV. Concurrent occurrence of sepsis was noticed in 17.3% of patients contributing to unfavourable outcomes. Significant association of recovery was found in patients presenting with lower NYHA class at admission, lower BNP levels, higher baseline EF, smaller LVEDD and lower PASP. Moreover, trends of recovery were better in older patients. Mean hospital stay (6.29 vs 8.35 days) and CARPREG II score (3.79 vs 5.39) of recovered patients was less as compared to non-recovered patients (p value 0.011 & 0.016 respectively).

- We compared PPCM with and without PIH patients and found that patients with PIH were older, had higher baseline EF, lower LVEDD and more rates of recovery but none of these findings were significant. Preterm birth was more common in PIH subgroup patients which was also a significant statistical finding. (p value 0.009)
- Among HDP patients, obesity and primiparity were prevalent risk factors for occurrence of the disease. Pre-eclampsia was the most observed (73.3%); preterm birth and low birth weight was prevalent in 57.8% and 48.1% of cases respectively; on echocardiography diastolic dysfunction was found in 37.8% of the patients but presence of diastolic dysfunction had no association with any outcomes in these patients.
- Maternal mortality was found in 8.14% of our subjects and it was significantly associated with severe baseline symptoms, post-partum diagnosis, presence of sepsis& PPH and low hemoglobin levels.

CONCLUSION:

In our region of the world, maternal mortality is a major problem that requires medical care. Multidisciplinary planning and the cardio-obstetrics team are essential for making the most of the resources at hand and providing pregnant cardiac patients with the right kind of treatment. All things considered, our research contributes to the expanding body of knowledge on various heart diseases in pregnant females and emphasizes the necessity of early detection and focused treatment approaches. Pregnancy outcomes for the mother and the unborn child are improved by preconception diagnosis, counseling, appropriate referral, routine prenatal monitoring, and delivery at a facility with the necessary equipment.

LIMITATIONS:

Our study had no control group which would have yielded better results. It was a single centered study done in a tertiary care hospital where sickest of patients are admitted; so, these trends might not be generalised to entire population. A longer follow up might yield more outcomes.

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S.No	Name	DIAGNOSIS	MRD NUMBER	D.O.A/ OPD DATE	Age at Conception	BOOKED/ UNBOOKED	ADMITTED AT HEART CENTRE	PRE LSCS/ VD	POST LSCS/ VD	POST PARTUM (DAYS)	POST PARTUM (DAYS) GP	Parity 1	ABOORTION	TWINNPREGNANCYES	ivf treatment	SHORTNESS OF BREATH
1	AY	PPCM	1530903	06-06-2023	31	UNBOOKED	NO	NO	YES	1	Y	P3L3	N	YES	NO	Y
2	NG	PPCM	1473446	01-03-2023	32	BOOKED	YES	NO	YES	1	Y	P3L3	N	NO	NO	Y
3	SK	PPCM	1447910	18-01-2023	35	UNBOOKED	NO	YES	NO		N	G2A1	Y	NO	NO	Y
4	TT	PPCM	1508579	01-05-2023	30	UNBOOKED	YES	NO	YES	1	Y	P2L2	N	NO	NO	Y
5	R	PPCM	1711098	03-06-2024	21	BOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	Y
6	TS	PPCM	1499820	13-04-2023	39	UNBOOKED	YES	NO	YES	1	Y	P3L3	N	NO	NO	Y
7	K	PPCM	1503438	21-04-2023	25	UNBOOKED	YES	NO	YES	1	Y	P2L2	N	NO	NO	Y
8	ES	PPCM	1753896	18-06-2024	29	UNBOOKED	NO	NO	YES	1	Y	P1L1	N	NO	NO	Y
9	N	PPCM	1536526	15-06-2023	36	UNBOOKED	YES	NO	YES	60	Y	P1L1	N	NO	NO	Y
10	S	PPCM	1652097	12-01-2024	29	UNBOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	Y
11	NK	PPCM	1774135	13-10-2023	32	BOOKED	YES	YES	NO		N	PRIMI	N	NO	NO	Y
12	K	PPCM	1448509	20-01-2023	25	UNBOOKED	NO	NO	YES	1	Y	P1L1	N	NO	NO	Y
13	SP	PPCM	385917	01-05-2024	30	UNBOOKED	NO	NO	YES	1	Y	P2L2A1	Y	NO	NO	Y
14	GK	PPCM	1773331	17-07-2024	32	BOOKED	NO	NO	YES	20	Y	P3L3	N	NO	NO	Y
15	NG	PPCM	1251072	23-08-2023	34	UNBOOKED	NO	NO	NO		N	G2P1L1	N	NO	NO	Y
16	IV	PPCM	1545468	01-07-2023	29	UNBOOKED	YES	NO	YES	1	Y	P1L1	N	NO	NO	Y
17	A	PPCM	1558965	25-07-2023	25	UNBOOKED	YES	NO	YES	1	Y	P1A1	Y	NO	NO	Y
18	V	PPCM	1572318	17-08-2023	24	BOOKED	YES	NO	YES	3	Y	P1L1	N	NO	NO	Y
19	V	PPCM	1581182	31-08-2023	19	UNBOOKED	NO	NO	NO		N	PRIMI	N	NO	NO	N
20	S	PPCM	1564052	03-08-2023	28	UNBOOKED	YES	NO	YES	20	Y	P1L1	N	NO	NO	Y
21	RR	PPCM	1296667	11-11-2023	34	UNBOOKED	YES	NO	YES	60	Y	P2L2	N	NO	NO	Y
22	MK	PPCM	1552473	14-07-2023	34	UNBOOKED	YES	NO	YES	1	Y	P2L2	N	NO	NO	Y
23	SK	PPCM	1541667	24-06-2023	29	UNBOOKED	YES	NO	YES	3	Y	P1L1	N	NO	NO	Y
24	PK	PPCM	1593613	20-09-2023	33	UNBOOKED	YES	NO	YES	1	Y	P2L2	N	YES	NO	Y
25	MK	PPCM	1578579	26-08-2023	28	UNBOOKED	YES	YES	NO		N	PRIMI	N	NO	NO	Y
26	P	PPCM	1600085	02-10-2023	28	BOOKED	YES	YES	NO		N	PRIMI	N	YES	NO	Y
27	PR	PPCM	1599474	29-09-2023	29	BOOKED	YES	NO	YES	1	Y	P1L1	N	NO	NO	Y
28	PF	PPCM	1619843	07-11-2023	30	UNBOOKED	NO	NO	YES	1	Y	PRIMI	N	NO	NO	Y
29	MK	PPCM	1703224	28-03-2024	32	UNBOOKED	YES	NO	YES	1	Y	P2L2	N	NO	NO	Y
30	SK	PPCM	397745	OPD 21-06-2023	29	BOOKED	YES	NO	YES	20	Y	P2L2	N	NO	NO	Y
31	SK	PPCM	1607884	14-10-2023	30	UNBOOKED	NO	NO	YES	1	Y	G4P3L3	N	NO	NO	Y
32	SK	PPCM	1625360	18-11-2023	32	BOOKED	NO	YES	NO		N	PRIMI	N	YES	NO	Y
33	RK	PPCM	1170035	06-10-2023	38	BOOKED	NO	NO	YES	5	Y	P1L1	N	YES	YES	Y
34	RD	PPCM	1734705	15-05-2024	43	UNBOOKED	NO	YES	NO		N	G4P2L1A1	Y	YES	NO	Y
35	SC	PPCM	1353668	05-02-2023	24	UNBOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	Y
36	S	PPCM	1575341	21-08-2023	37	BOOKED	YES	YES	NO		N	G2P1L1	N	NO	NO	Y
37	AK	PPCM	1574419	19-08-2023	42	UNBOOKED	YES	NO	YES	1	Y	P3L3	N	NO	NO	Y
38	LK	PPCM	1697807	29-11-2023	38	UNBOOKED	YES	NO	YES	1	Y	P2L1A1	Y	NO	NO	Y
39	SK	PPCM	730479	OPD 03-06-2023	32	UNBOOKED	NO	NO	YES	60	Y	P3L3	N	NO	NO	Y
40	JK	PPCM	1497615	11-04-2023	42	UNBOOKED	NO	NO	YES	1	Y	P2L1A1	Y	NO	YES	Y
41	PS	PPCM	1302895	26-02-2023	29	BOOKED	YES	YES	NO		N	G3P1L1A1	Y	NO	NO	N
42	P	PPCM	1488590	27-03-2023	34	UNBOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	Y
43	A	PPCM	1469012	24-02-2023	26	UNBOOKED	NO	NO	YES	1	Y	P2L2	N	NO	NO	Y
44	MK	PPCM	1546961	04-07-2023	29	UNBOOKED	NO	NO	YES	8	Y	P2L2	N	NO	NO	Y
45	GK	PPCM	1652866	13-01-2024	31	BOOKED	NO	YES	NO		N	G2P1L0	N	NO	NO	Y
46	A	PPCM	1684936	02-03-2024	28	UNBOOKED	NO	YES	NO		N	G2P1L1	N	NO	NO	Y
47	PK	PPCM	1720813	24-04-2024	33	UNBOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	Y
48	D	PPCM	1732161	17-05-2024	27	UNBOOKED	NO	NO	YES	1	Y	P1A1	Y	NO	NO	Y
49	RK	PPCM	1738158	22-05-2024	34	UNBOOKED	NO	NO	YES	1	Y	P2L1A1	Y	NO	NO	Y
50	CK	PPCM	1197368	16-06-2024	37	UNBOOKED	NO	NO	YES	1	Y	P2L2	N	NO	NO	Y
51	SK	PPCM	1664581	01-02-2024	30	BOOKED	NO	NO	YES	2	Y	P1L1	N	NO	NO	Y

S.No	PALPITATIONS	GTCS	INCREASED BP READINGS	FEVER	POST PARTUM HEMORRHAGE	INCREASED SUGARS	SHOCK	OTHERS	DURATION OF SYMPTOMS (DAYS) GP	NYHA CLASS (ADMSN)	BMI	PREGNANCY DURATION (TERMINATION)	mode of delievery
1	N	N	Y	N	N	N	N	N	1	IV	27	34.0	LSCS
2	N	N	Y	N	N	N	N	N	1	III	23	35.0	LSCS
3	N	N	Y	N	N	N	N	N	1	III	24	36.1	LSCS
4	N	N	N	N	N	N	N	N	1	III	24	37.0	LSCS
5	N	N	N	N	N	N	N	N	1	III	24	36.0	LSCS
6	N	N	N	N	N	N	N	N	1	III	29	35.0	LSCS
7	N	N	N	N	N	N	N	N	1	IV	24	35.0	LSCS
8	N	N	Y	N	N	N	N	N	1	III	27	37.0	VAGINAL DELIEVERY
9	N	N	N	N	N	N	N	N	2	III	27	38.0	LSCS
10	N	N	Y	N	N	N	N	N	1	II	22	38.4	LSCS
11	N	N	Y	N	N	N	N	N	1	III	25	35.0	LSCS
12	N	N	N	N	N	N	N	N	1	III	29	35.0	VAGINAL DELIEVERY
13	N	N	N	N	N	N	N	N	1	II	24	37.0	VAGINAL DELIEVERY
14	N	N	Y	N	N	N	N	N	1	IV	28	34.0	LSCS
15	N	N	N	N	N	N	N	N	1	II	27	35.0	LSCS
16	N	N	Y	N	N	N	N	N	1	IV	24	34.0	LSCS
17	N	N	Y	N	N	N	N	N	1	IV	23	27.0	LSCS
18	Y	N	N	N	N	N	N	N	1	II	26	34.0	LSCS
19	N	Y	N	N	N	N	N	N	1	NA	23	28.0	VAGINAL DELIEVERY
20	Y	N	N	N	N	N	N	N	1	IV	28	37.0	LSCS
21	N	N	Y	N	N	N	N	N	1	III	27	37.0	VAGINAL DELIEVERY
22	N	N	N	N	N	N	Y	N	2	IV	31	34.0	LSCS
23	N	N	N	N	N	N	N	PRESYNCOPE	1	III	24	37.0	LSCS
24	N	N	N	N	Y	N	Y	N	1	IV	27	37.0	LSCS
25	N	N	N	Y	N	N	N	N	1	II	31	33.4	VAGINAL DELIEVERY
26	N	N	Y	N	N	N	N	N	1	III	27	33.0	LSCS
27	N	N	Y	N	N	N	N	N	1	III	26	36.0	LSCS
28	N	N	N	N	N	N	N	N	1	IV	27	31.0	LSCS
29	N	N	Y	N	N	N	N	N	2	III	28	38.0	LSCS
30	N	N	Y	N	N	N	N	N	2	II	29	35.0	LSCS
31	N	N	N	N	N	N	N	N	1	III	31	34.0	LSCS
32	N	N	N	N	N	N	N	N	1	II	24	33.0	LSCS
33	N	N	N	N	N	N	N	N	2	III	32	36.0	LSCS
34	N	N	Y	N	N	N	N	N	2	II	28	29.6	LSCS
35	N	N	N	N	N	N	N	N	1	III	25	38.0	LSCS
36	N	N	N	N	N	N	N	CHEST PAIN	2	II	29	37.2	LSCS
37	N	N	Y	N	N	N	N	N	1	III	29	36.0	LSCS
38	N	N	Y	N	N	N	N	N	1	II	29	36.1	LSCS
39	N	N	Y	N	N	N	N	N	1	II	28	34.0	VAGINAL DELIEVERY
40	N	N	Y	N	N	N	N	N	1	II	30	38.0	LSCS
41	N	N	N	N	N	N	N	N		NA	26	37.0	LSCS
42	N	N	Y	N	N	N	N	N	1	IV	29	35.3	LSCS
43	N	N	N	N	N	N	N	N	1	IV	28	34.0	LSCS
44	N	N	N	N	N	N	Y	N	1	IV	24	35.0	LSCS
45	N	N	Y	N	N	N	N	N	1	IV	28	34.1	VAGINAL DELIEVERY
46	N	N	N	Y	N	N	N	N	1	III	26	15.2	VAGINAL DELIEVERY
47	N	N	N	N	N	N	Y	N	1	IV	29	16.2	VAGINAL DELIEVERY
48	N	N	N	N	N	N	N	N	1	IV	25	25.0	VAGINAL DELIEVERY
49	N	N	N	N	N	N	Y	N	1	IV	27	33.0	LSCS
50	N	N	N	N	Y	N	Y	N	1	IV	24	34.0	VAGINAL DELIEVERY
51	N	N	Y	N	N	N	N	N	1	II	27	35.2	LSCS

S.No	HYPERTENSION	BRONCHIAL ASTHMA	HYPOTHYROIDISM	HYPERTHYROIDISM	DM	H/O STROKE	K/C/O CHD	K/C/O RHD	HISTORY OF DVT	OTHERS	CARDIAC Surgery before pregnancy	ECG	QRS	Echo	EF (%)
1	Y	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	SEVERE GHK OF LV	20
2	N	Y	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA; ST DEPRESSION INFERIOR LEADS	85	SEVERE LV SYSTOLIC DYSFUNCTION	25
3	N	N	N	N	N	N	N	N	N		NONE	SINUS BRADYCARDIA	96	GHK OF LV	45
4	N	N	N	N	N	N	N	N	N		NONE	SVT, FREQUENT VENTRICULAR ECTOPICS	100	SEVERE GHK OF LV, MILD MR	20
5	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	88	SEVERE GHK OF LV	22
6	N	N	Y	N	N	N	N	N	N		NONE	NORMAL SINUS RHYTHM	80	GHK OF LV, EF 35%	35
7	N	N	N	N	Y	N	N	N	N		NONE	SINUS TACHYCARDIA, LVH	110	SEVERE GHK OF LV, SEVERE MR	16
8	Y	N	N	N	N	N	N	N	N		NONE	NORMAL SINUS RHYTHM, FREQUENT VPC'S	115	GHK OF LV , MOD MR	36
9	Y	N	N	N	N	N	N	N	N		NONE	ATRIAL TACHYCARDIA, T WAVE	95	SEVERE GHK OF LV	25
10	Y	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	100	CONCENTRIC LVH WITH GHK OF LV	42
11	N	N	Y	N	Y	N	N	N	N		NONE	NSR	90	GHK OF LV	25
12	N	N	Y	N	N	N	N	N	N		NONE	NSR	85	GHK OF LV	42
13	N	N	N	N	N	N	N	N	N		NONE	NSR	90	GHK OF LV	44
14	Y	N	N	N	N	N	N	N	N		NONE	ATRIAL FIBRILLATION	110	SEVERE GLOBAL HYPOKINESIA OF LV	25
15	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	96	GHK OF LV	45
16	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	100	SEVERE GHK OF LV	18
17	N	N	Y	N	N	N	N	N	N		NONE	SINUS TACHY, T INVERSION V1-V6	120	GHK OF LV EF 25%	25
18	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA , SHORT PR INTERVAL; SVT(SHORT RP) ?WPW	100	SEVERE GHK OF LV ,MOD MR	28
19	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	100	GHK OF LV , EF 32%	32
20	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	GHK OF LV , MOD MR	20
21	Y	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	100	GHK OF LV , EF 38%	38
22	N	N	Y	N	N	N	N	N	N		NONE	NSR, QS IN V1-V4, MULTIFOCAL	110	SEVERE GHK OF LV,MODERATE MR	14
23	N	N	N	N	N	N	N	N	N		NONE	INTERMITTENT JUNCTIONAL RHYTHM AND AV BLOCKS	100	SEVERE GHK OF LV	20
24	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA, IVCD+	125	MILD GHK OF LV , EF 42%	42
25	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	82	MODERATE LV SYSTOLIC DYSFUNCTION , EF 32%	32
26	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	MODERATE LV SYSTOLIC DYSFUNCTION , MOD MR	38
27	N	N	N	N	N	N	N	N	N		NONE	NSR	90	SEVERE GHK OF LV ,MOD PERICARDIAL	26
28	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	124	SEVERE GHK OF LV ,	26
29	N	N	N	N	N	N	N	N	N		NONE	NSR	110	GHK OF LV, EF 26-28%	26
30	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	120	GHK OF LV EF 40%	40
31	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	90	GHK OF LV , EF 34%	34
32	Y	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	GHK OF LV , EF 30%, LA MODERATELY	30
33	N	N	N	N	Y	N	N	N	N		NONE	SINUS TACHYCARDIA	100	GHK OF LV , EF 38%	38
34	Y	N	N	N	N	N	N	N	N		NONE	NSR	100	GHK OF LV ,MODERTAE TR	40
35	N	N	N	N	N	N	N	N	N		NONE	NSR	90	MILD GHK OF LV, EF 42%	42
36	N	N	N	N	N	N	N	N	N		NONE	LBBB, SHORT PR INTERVAL	130	GHK OF LV, EF 40%	40
37	Y	N	Y	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA, LVH	90	MILD GHK OF LV	42
38	N	N	Y	N	N	N	N	N	N		NONE	SINUS BRADYCARDIA WITH PVC'S	96	GHK OF LV , EF 40%	40
39	N	N	N	N	N	N	N	N	N		NONE	NSR	110	GHK OF LV , EF 42%	42
40	Y	N	N	N	N	N	N	N	N		NONE	NSR	120	GHK OF LV	35
41	N	N	Y	N	N	N	N	N	N	H/O PPCM	NONE	SINUS TACHYCARDIA	100	NORMAL LVEF	60
42	Y	N	N	Y	N	N	N	N	N		NONE	SINUS TACHYCARDIA, IVCD	128	SEVERE GHK OF LV	22
43	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	106	SEVERE GHK OF LV , EF 10-12%; SEVERE RV	10
44	N	N	N	N	N	N	N	N	N		NONE	POLYMORPHIC VT, PROLONGED	138	GHK OF LV , RA/RV MODERATELY DILATED	35
45	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	SEVERE GHK OF LV ,MOD MR	26
46	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	120	GHK OF LV , EF 32%	32
47	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	128	SEVERE GHK OF LV ,MILD PERICARDIAL	28
48	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	100	VEGETATION ON STL , MODERATE TR,	25
49	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	94	SEVERE GHK OF LV; MILD TO MODERATE	10
50	Y	N	N	N	N	N	N	N	N		NONE	NSR	90	SEVERE GHK OF LV , MILD TR	14
51	N	N	N	N	N	N	N	N	N		NONE	NSR	100	GHK OF LV	42

S.No	RV DISFUNCTION IF YES RVFAC	RVFAC	PASP	LVH	LVEDD	LVESD	DIASTOLIC DYSFUNCTION	DIASTOLIC DYSFUNCTION TYPE	HEMOGLOBIN	BNP	Congenital HD	Acquired HD	GDMA2	PIH	GESTATIONAL HTN	PRE ECLAMPSIA
1	YES	20	40	NO	48	29	YES	TYPE 2	12	3610	NONE	PPCM	N	Y	N	Y
2	NO	35	30	NO	43	28	NO		13.2	608	NONE	PPCM	Y	Y	N	Y
3	NO	35	25	NO	52	31	NO		9.2	190	NONE	PPCM	N	Y	N	Y
4	NO	35	45	NO	53	43	NO		10.7	870	MVP	PPCM	N	N	N	N
5	NO	35	40	NO	56	46	NO		12.5	915	NONE	PPCM	N	N	N	N
6	NO	35	35	NO	58	27	NO		12.6	685	NONE	PPCM	N	N	N	N
7	NO	35	50	NO	62	44	YES	TYPE 3	9.8	1600	NONE	PPCM	N	N	N	N
8	NO	35	48	NO	53	39	YES	TYPE 2	13.5	1700	NONE	PPCM	N	Y	N	N
9	NO	35	50	NO	62	42	NO		14.5	1230	NONE	PPCM	N	N	N	N
10	NO	35	20	YES,MODERATE	50	30	NO		11.3	660	NONE	PPCM	N	Y	N	N
11	NO	35	28	NO	53	36	NO		13.5	1800	NONE	PPCM	N	Y	N	N
12	NO	35	20	NO	59	45	NO		8.3	775	NONE	PPCM	N	N	N	N
13	NO	35	25	NO	56	42	NO		13.5	455	NONE	PPCM	N	N	N	N
14	NO	35	25	NO	53	37	NO		9.3	1200	NONE	PPCM	N	Y	N	Y
15	NO	35	20	NO	48	36	NO		10.5	350	NONE	PPCM	N	N	N	N
16	YES	28	36	NO	59	44	YES	TYPE 2	13.1	1800	NONE	PPCM	N	N	N	N
17	NO	35	55	NO	64	48	NO		11.8	5000	NONE	PPCM	N	Y	N	N
18	NO	35	45	NO	60	44	NO		12.5	650	NONE	PPCM	N	N	N	N
19	NO	35	25	NO	59	41	NO		8.4	350	NONE	PPCM	N	Y	N	N
20	NO	35	48	NO	60	39	YES	TYPE 3	9.5	3100	NONE	PPCM	N	N	N	N
21	NO	35	30	NO	52	38	NO		13.2	650	NONE	PPCM	N	Y	N	Y
22	NO	35	50	NO	63	43	YES	TYPE 2	13	925	NONE	PPCM	N	N	N	N
23	NO	35	40	NO	54	39	YES	TYPE 1	12	1310	NONE	PPCM	N	N	N	N
24	NO	35	20	NO	54	35	NO		8.5	230	NONE	PPCM	N	N	N	N
25	NO	35	25	NO	56	37	NO		7.9	400	NONE	PPCM	N	N	N	N
26	NO	35	45	NO	53	35	YES	TYPE 2	9.3	540	NONE	PPCM	N	Y	N	Y
27	NO	35	50	NO	61	49	YES	TYPE 3	7.4	3200	NONE	PPCM	N	Y	N	Y
28	NO	35	42	NO	63	40	YES	TYPE 2	10.5	2200	NONE	PPCM	N	N	N	N
29	NO	35	35	NO	58	44	NO		10.4	800	NONE	PPCM	N	Y	N	Y
30	NO	35	28	NO	51	40	NO		12.2	245	NONE	PPCM	N	Y	N	Y
31	NO	35	25	NO	56	32	NO		12.2	400	NONE	PPCM	N	N	N	N
32	NO	35	30	NO	46	26	YES	TYPE 2	13.5	835	NONE	PPCM	N	N	N	N
33	NO	35	25	NO	52	34	NO		12.4	455	NONE	PPCM	N	N	N	N
34	YES	25	55	NO	52	38	YES	TYPE 2	11.7	1100	NONE	PPCM	N	Y	N	Y
35	NO	35	18	NO	44	34	NO		8.5	175	NONE	PPCM	Y	N	N	N
36	NO	35	15	NO	51	35	NO		10.2	350	NONE	PPCM	N	N	N	N
37	NO	35	20	NO	56	37	NO		12.4	310	NONE	PPCM	Y	Y	N	N
38	NO	35	20	NO	47	26	NO		13.5	240	NONE	PPCM	N	Y	N	Y
39	NO	35	15	NO	48	34	NO		12.6	400	NONE	PPCM	N	Y	N	Y
40	NO	35	18	NO	50	32	YES	TYPE 2	12.4	800	NONE	PPCM	N	Y	N	N
41	NO	35	15	NO	41	27	NO		12	5	NONE	PPCM	Y	N	N	N
42	YES	14	45	NO	62	40	YES	TYPE 3	13.1	1920	NONE	PPCM	N	Y	N	N
43	YES	8	50	NO	55	45	YES	TYPE 2	4.6	3200	NONE	PPCM	N	N	N	N
44	YES	22	60	NO	59	42	YES	TYPE 3	6.5	1490	NONE	PPCM	N	N	N	N
45	NO	35	50	NO	54	44	YES	TYPE 2	11.6	2300	NONE	PPCM	N	Y	N	N
46	NO	35	30	NO	48	39	YES	TYPE 2	9.4	600	NONE	PPCM	N	N	N	N
47	NO	35	35	NO	45	34	NO		11.2	1800	NONE	PPCM	N	N	N	N
48	YES	8	50	NO	58	50	NO		10.4	1500	NONE	PPCM	N	N	N	N
49	NO	35	40	NO	59	48	NO		7.2	3100	NONE	PPCM	N	N	N	N
50	YES	12	45	NO	59	47	YES	TYPE 2	8.5	1200	NONE	PPCM	N	N	N	N
51	NO	35	20	NO	44	25	NO		12.5	400	NONE	PPCM	N	Y	N	Y

S.No	PRE ECLAMPSIA WITH SEVERE FEATURES	ECLAMPSIA	SEPSIS	AKI	VTE	OTHERS	FGR	MACROSMIA	ANHYDROAMNIOS	OLIGOHYDROAMNIOS	POLYHYDRAMNIOS	PPH	PRE TERM	PPROM	FETAL/ PERINATAL COMPLICATIONS	LOW BIRTH WEIGHT (GM)
1	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
2	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
3	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
4	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
5	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
6	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
7	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
8	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
9	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
10	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1280
11	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1600
12	N	N	Y	N	N	N	N	N	N	N	N	Y	Y	N	LOW BIRTH WEIGHT	1300
13	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
14	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	NONE	
15	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
16	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1230
17	Y	N	Y	Y	N	N	Y	N	N	N	N	N	Y	N	IUD	
18	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
19	N	Y	N	N	N	PRES	N	N	N	N	N	N	Y	Y	IUD	
20	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
21	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
22	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1600
23	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	NONE	
24	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	NONE	
25	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	IUD	
26	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	A-2020,B-1680
27	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
28	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1320
29	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
30	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1600
31	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1800
32	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1700
33	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	A-1900,B-1800
34	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LBW (FETUS A), IUD (FETUS B)	
35	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	NONE	
36	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
37	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
38	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
39	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	NONE	
40	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1630
41	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
42	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	IUD	
43	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	NONE	
44	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	NONE	
45	Y	N	N	N	N	N	Y	N	Y	N	N	N	N	N	IUD	
46	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	IUD	
47	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	IUD	
48	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	IUD	
49	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	IUD	
50	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
51	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	

S.No	PRIMARY OUTCOMES	MECHANICAL VENTILATOR	BETA BLOCKERS	RAAS #	LOOP DIURETICS	MRA	ANTICOAGULATION	OTHERS	HOSPITAL STAY (DAYS)	CARPREG II SCORING	6mn FOLLOW UP NYHA	EF AT 6 MONTHS	RECOVERED	MORATILTY
1	LEFT VENTRICULAR FAILURE	Y	Y	Y,ACE-I	N	N	N	N	7	5	II	25	NO	N
2	LEFT VENTRICULAR FAILURE	N	Y	Y,ARNI	Y	Y	N	N	5	6	I	60	YES	N
3	LEFT VENTRICULAR FAILURE	N	Y	Y,ACE-I	N	N	N	N	6	3	I	60	YES	N
4	LEFT VENTRICULAR FAILURE	Y	Y	Y,ACE-I	Y	Y	N	N	6	6	II	60	YES	N
5	LEFT VENTRICULAR FAILURE	N	N	N	N	Y	N	IVABRADINE	6	5	II	25	NO	N
6	LEFT VENTRICULAR FAILURE	N	Y	Y,ACE-I	Y	Y	N	N	6	6	I	60	YES	N
7	LEFT VENTRICULAR FAILURE	N	Y	Y,ACE-I	N	Y	N	N	7	8	I	42	NO	N
8	LEFT VENTRICULAR FAILURE	N	Y	Y,ARNI	N	N	N	N	6	7	I	55	YES	N
9	LEFT VENTRICULAR FAILURE	N	Y	Y,ARB	Y	Y	N	N	9	7	II	30	NO	N
10	LEFT VENTRICULAR FAILURE	N	Y	N	N	Y	N	N	7	2	I	55	YES	N
11	LEFT VENTRICULAR FAILURE	N	Y	Y,ARB	Y	Y	N	N	5	5	II	40	NO	N
12	LEFT VENTRICULAR FAILURE	N	Y	Y,ACE-I	N	N	N	N	7	6	I	55	YES	N
13	LEFT VENTRICULAR FAILURE	N	Y	N	N	Y	N	N	5	2	I	60	NO	N
14	LEFT VENTRICULAR FAILURE	N	Y	Y,ARB	Y	Y	N	N	8	5	I	60	NO	N
15	LEFT VENTRICULAR FAILURE	N	Y	N	N	N	N	N	5	2	I	60	NO	N
16	LEFT VENTRICULAR FAILURE	Y	Y	N	N	Y	N	N	7	8	II	35	NO	N
17	LEFT VENTRICULAR FAILURE	Y	N	N	Y	Y	N	HYDRALAZINE,IVABRADINE	11	8	II	30	NO	N
18	LEFT VENTRICULAR FAILURE	N	Y	Y,ARNI	N	N	N	SGLT2#	9	4	I	56	YES	N
19	LEFT VENTRICULAR FAILURE	N	Y	N	N	N	N	N	12	2	II	30	NO	N
20	LEFT VENTRICULAR FAILURE	Y	N	Y,ACE-I	Y	Y	N	SGLT2#	8	8	II	40	NO	N
21	LEFT VENTRICULAR FAILURE	N	Y	Y,ARB	N	Y	N	N	4	5	I	50	YES	N
22	LEFT VENTRICULAR FAILURE	Y	Y	N	Y	Y	N	N	10	8	II	28	NO	N
23	LEFT VENTRICULAR FAILURE	N	N	N	N	Y	N	IVABRADINE	15	5	I	60	YES	N
24	LEFT VENTRICULAR FAILURE	Y	N	N	N	N	N	IVABRADINE	7	4	I	60	YES	N
25	LEFT VENTRICULAR FAILURE	N	N	Y,ACE-I	N	N	N	N	5	3	I	55	YES	N
26	LEFT VENTRICULAR FAILURE	N	Y	Y,ARB	N	N	N	N	5	4	I	55	YES	N
27	LEFT VENTRICULAR FAILURE	N	Y	Y,ARB	Y	Y	N	N	7	8	II	30	NO	N
28	LEFT VENTRICULAR FAILURE	N	N	N	N	N	N	N	14	7	II	30	NO	N
29	LEFT VENTRICULAR FAILURE	N	Y	N	N	N	N	SGLT2#	8	5	II	60	YES	N
30	LEFT VENTRICULAR FAILURE	N	Y	N	N	Y	N	N		2	II	45	NO	N
31	LEFT VENTRICULAR FAILURE	Y	N	N	N	N	N	N	17	5	MATERNAL DEATH			Y
32	LEFT VENTRICULAR FAILURE	N	N	N	N	N	N	N	7	2	I	50	YES	N
33	LEFT VENTRICULAR FAILURE	N	Y	Y,ARNI	N	Y	N	N	5	2	I	60	YES	N
34	BIVENTRICULAR FAILURE	N	N	N	N	N	N	N	12	4	II	40	NO	N
35	LEFT VENTRICULAR FAILURE	N	Y	N	N	Y	N	INSULIN	4	2	I	60	YES	N
36	LEFT VENTRICULAR FAILURE	N	N	Y,ACE-I	N	N	N	IVABRADINE	4	2	I	55	YES	N
37	LEFT VENTRICULAR FAILURE	N	Y	Y,ARNI	N	N	N	N	6	5	I	60	YES	N
38	LEFT VENTRICULAR FAILURE	N	Y	Y,ACE-I	N	N	N	N	6	3	I	60	YES	N
39	LEFT VENTRICULAR FAILURE	N	Y	Y,ARB	N	Y	N	N	5	2	I	55	YES	N
40	NONE	N	Y	N	N	Y	N	N	7	3	I	60	YES	N
41	NONE	N	Y	N	N	N	N	INSULIN	5	2	I	60	YES	N
42	MATERNAL DEATH	N	N	N	N	N	N	N	1	7	MATERNAL DEATH			Y
43	MATERNAL DEATH	N	N	N	N	N	N	N	1	7	MATERNAL DEATH			Y
44	MATERNAL DEATH	N	N	N	N	N	N	N	1	7	MATERNAL DEATH			Y
45	MATERNAL DEATH	N	N	N	N	N	N	N	2	7	MATERNAL DEATH			Y
46	MATERNAL DEATH	N	N	N	N	N	N	N	3	2	MATERNAL DEATH			Y
47	MATERNAL DEATH	N	N	N	N	N	N	N	2	5	MATERNAL DEATH			Y
48	MATERNAL DEATH	N	N	N	N	N	N	N	2	7	MATERNAL DEATH			Y
49	MATERNAL DEATH	N	N	N	N	N	N	N	3	7	MATERNAL DEATH			Y
50	MATERNAL DEATH	N	N	N	N	N	N	N	2	7	MATERNAL DEATH			Y
51	LEFT VENTRICULAR FAILURE	N	Y	N	N	Y	N	N	6	2	I	60	YES	N

S.No	Name	DIAGNOSIS	MRD NUMBER	D.O.A/ OPD DATE	Age at Conception	BOOKED/ UNBOOKED	ADMITTED AT HEART CENTRE	PRE LSCS/ VD	POST LSCS/ VD	POST PARTUM (DAYS)	POST PARTUM (DAYS) GP	Parity 1	ABOORTION	TWINNPREGNANCYES	ivf treatment	SHORTNESS OF BREATH
52	HK	PPCM	1084340	10-04-2023	30	BOOKED	NO	NO	NO		N	G2A1	Y	NO	NO	Y
53	S	HDP	1628818	07-01-2024	33	BOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	N
54	NP	HDP	1746012	04-06-2024	27	UNBOOKED	NO	NO	YES		N	PRIMI	N	NO	NO	N
55	M	HDP	1649383	06-01-2024	30	UNBOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	Y
56	B	HDP	1290474	30-10-2023	48	UNBOOKED	NO	NO	YES		N	G2A1	Y	NO	NO	N
57	K	HDP	1440202	08-01-2023	31	BOOKED	NO	NO	YES	5	Y	P1L1	N	NO	NO	N
58	M	HDP	1440358	03-01-2023	29	UNBOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	N
59	P	HDP	1368458	07-01-2023	32	UNBOOKED	NO	NO	YES		N	PRIMI(TWIN PREGNANCY)	N	YES	YES	N
60	M	HDP	1615055	28-10-2023	38	BOOKED	NO	NO	YES		N	G2A1	Y	NO	NO	N
61	D	HDP	584316	14-10-2023	32	UNBOOKED	NO	NO	YES		N	G3P1L1A1	Y	NO	NO	N
62	MK	HDP	1659353	25-01-2024	25	UNBOOKED	NO	NO	YES		N	PRIMI	N	NO	NO	N
63	S	HDP	1446759	18-01-2023	44	BOOKED	NO	NO	YES		N	PRIMI(TWIN PREGNANCY)	N	YES	YES	N
64	SS	HDP	1449778	23-01-2023	34	UNBOOKED	NO	NO	YES		N	G2P1L1(TWIN PREGNANCY)	N	YES	NO	N
65	JK	HDP	1497615	11-04-2023	42	BOOKED	NO	NO	YES		N	G2A1	Y	NO	YES	N
66	HK	HDP	1547394	14-07-2023	44	BOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	N
67	J	HDP	1762684	29-11-2023	26	UNBOOKED	NO	NO	YES		N	PRIMI	N	NO	NO	N
68	JK	HDP	1495298	08-04-2023	28	UNBOOKED	NO	NO	YES		N	G2A1	Y	NO	NO	N
69	R	HDP	1540523	23-06-2023	34	BOOKED	NO	NO	YES		N	G3A2	Y	NO	NO	N
70	BK	HDP	1615415	30-10-2023	42	UNBOOKED	NO	NO	YES		N	PRIMI(TWIN PREGNANCY)	N	YES	YES	N
71	RK	HDP	1493930	05-04-2023	36	BOOKED	NO	NO	YES		N	G3P1L1A1	Y	NO	NO	N
72	KK	HDP	1443547	18-03-2023	44	UNBOOKED	NO	NO	YES		N	PRIMI(TWIN PREGNANCY)	N	YES	NO	N
73	M	HDP	1477567	OPD 20-07-2023	25	UNBOOKED	NO	NO	YES	90	Y	P1L1	N	NO	NO	N
74	NM	HDP	1537296	03-07-2023	40	UNBOOKED	NO	NO	YES		N	PRIMI	N	NO	NO	Y
75	SR	HDP	1476553	OPD 07-05-2023	28	BOOKED	NO	NO	YES	120	Y	P1L1	N	NO	NO	N
76	BR	HDP	7541	14-04-2023	40	UNBOOKED	NO	NO	YES		N	G3A2	Y	NO	YES	N
77	SR	HDP	1510430	OPD 03-05-2023	37	UNBOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	N
78	GK	HDP	1532130	OPD 15-06-2024	30	UNBOOKED	NO	NO	YES	120	Y	P1L1	N	NO	NO	N
79	N	HDP	1165969	03-01-2023	32	UNBOOKED	NO	NO	YES		N	PRIMI	N	NO	NO	N
80	RS	HDP	1441467	04-01-2023	35	UNBOOKED	NO	NO	YES		N	G2A1	Y	NO	NO	N
81	P	HDP	1368458	06-01-2023	32	BOOKED	NO	NO	YES		N	PRIMI (TWIN PREGNANCY)	N	YES	YES	N
82	Y	HDP	1564451	09-08-2023	32	UNBOOKED	NO	NO	YES		N	PRIMI	N	NO	NO	N
83	SD	HDP	1446759	16-01-2023	44	BOOKED	NO	YES	NO		N	PRIMI (TWIN PREGNANCY)	N	YES	YES	N
84	SK	HDP	1630508	OPD 29-11-2023	38	UNBOOKED	NO	YES	NO		N	G2P1L1	N	NO	NO	N
85	PK	HDP	1491650	01-04-2023	45	UNBOOKED	NO	NO	YES		N	G4P1L1A2	Y	NO	NO	N
86	SL	HDP	1503291	22-04-2023	30	UNBOOKED	NO	NO	YES		N	PRIMI	N	NO	NO	N
87	S	HDP	1426383	19-01-2023	19	UNBOOKED	NO	NO	YES	1	Y	PRIMI	N	NO	NO	N
88	P	HDP	1539863	19-12-2023	29	UNBOOKED	NO	NO	YES	2	Y	P2L2	N	NO	NO	N
89	CG	HDP	1619712	06-11-2023	29	BOOKED	NO	NO	YES	2	Y	P1L1	N	NO	NO	N
90	PK	HDP	1618696	04-11-2023	35	BOOKED	YES	NO	YES	1	Y	P2L2	N	NO	NO	N
91	DJ	HDP	47579	29-12-2023	37	BOOKED	NO	NO	YES		N	G2A1	Y	NO	NO	N
92	RK	HDP	1514730	24-12-2023	32	UNBOOKED	NO	NO	YES		N	G3P1L1A1	Y	NO	NO	N
93	ST	HDP	1643649	25-12-2023	35	UNBOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	N
94	SD	HDP	1614191	18-12-2023	38	BOOKED	NO	NO	YES		N	G3P2L2	N	NO	NO	N
95	S	HDP	1662880	04-02-2024	34	BOOKED	NO	NO	YES		N	PRIMI	N	NO	NO	N
96	C	HDP	1584602	06-09-2023	35	UNBOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	N
97	ST	HDP	823585	11-01-2023	35	UNBOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	N
98	M(	CHD	1617931	22-04-2024	22	UNBOOKED	NO	NO	YES		N	G2P1L1	N	NO	NO	N
99	HK	CHD	1557205	22-07-2023	30	BOOKED	YES	YES	NO		N	PRIMI	N	NO	NO	N
100	CK	CHD	1661351	28-01-2024	33	UNBOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	Y
101	P	CHD	1579272	28-08-2023	29	BOOKED	YES	YES	NO		N	PRIMI	N	NO	NO	N
102	RK	CHD	354135	17-05-2023	28	UNBOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	N
103	RK	CHD	8363	16-01-2023	34	BOOKED	NO	YES	NO		N	G2P1L1	N	NO	NO	N
104	JK	CHD	1511745	05-05-2023	30	UNBOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	N
105	PG	CHD	1626927	21-11-2023	32	BOOKED	YES	YES	NO		N	PRIMI	N	NO	NO	N
106	HK	CHD	1785337	05-05-2024	31	UNBOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	N
107	KK	VHD	1778289	13-08-2023	28	UNBOOKED	NO	NO	NO		N	G2P1L1	N	NO	NO	Y
108	AK	VHD	1465877	14-03-2023	32	BOOKED	YES	YES	NO		N	G3P1A2L0	Y	NO	NO	Y
109	KK	VHD	1555310	09-09-2023	37	UNBOOKED	NO	NO	NO		N	G2P1L1	N	NO	NO	N
110	KK	VHD	1002171	27-02-2023	37	UNBOOKED	NO	NO	YES	1	Y	G1A1	Y	NO	NO	N
111	AK	VHD	1613056	24-10-2023	32	BOOKED	YES	NO	YES	10	Y	P2L2	N	NO	NO	N

S.No	PALPITATIONS	GTCS	INCREASED BP READINGS	FEVER	POST PARTUM HEMORRHAGE	INCREASED SUGARS	SHOCK	OTHERS	DURATION OF SYMPTOMS (DAYS) GP	NYHA CLASS (ADMSN)	BMI	PREGNANCY DURATION (TERMINATION)	mode of delievery
52	N	N	N	N	N	N	N	N	1	III	27	34.3	LSCS
53	N	N	Y	N	N	N	N	N		NA	30	29.0	VAGINAL DELIEVERY
54	N	N	Y	N	N	N	N	N		NA	24	29.2	LSCS
55	N	N	N	N	N	N	N	N	1	II	27	33.4	LSCS
56	N	N	Y	N	N	N	N	N		NA	29	33.3	LSCS
57	N	N	Y	N	N	N	N	N	1	NA	28	36.0	LSCS
58	N	N	Y	N	N	N	N	N	2	NA	27	37.0	VAGINAL DELIEVERY
59	N	N	Y	N	N	N	N	N	2	NA	29	34.5	LSCS
60	N	N	Y	N	N	N	N	N		NA	30	33.3	LSCS
61	N	N	Y	N	N	N	N	N	2	NA	28	35.2	LSCS
62	N	N	Y	N	N	N	N	N	2	NA	23	35.3	LSCS
63	N	N	Y	N	N	N	N	N	2	NA	29	35.6	LSCS
64	N	N	Y	N	N	N	N	N	1	NA	27	34.5	LSCS
65	N	N	Y	N	N	N	N	N	2	NA	27	31.1	LSCS
66	N	N	Y	N	N	N	N	N	2	NA	30	24.0	VAGINAL DELIEVERY
67	N	N	Y	N	N	N	N	N	1	NA	24	37.0	LSCS
68	N	N	Y	N	N	N	N	N	2	NA	23	35.0	LSCS
69	N	N	Y	N	N	N	N	N	1	NA	26	37.0	LSCS
70	N	N	Y	N	N	N	N	N	2	NA	29	33.5	LSCS
71	N	N	Y	N	N	N	N	N	1	NA	26	31.5	LSCS
72	N	N	Y	N	N	N	N	N	1	NA	32	34.2	LSCS
73	N	N	Y	N	N	N	N	N	2	NA	28	37.0	VAGINAL DELIEVERY
74	N	N	N	N	N	N	N	N	1	II	31	32.3	LSCS
75	N	N	Y	N	N	N	N	N	1	NA	29	34.0	LSCS
76	N	N	Y	N	N	N	N	N	2	NA	26	37.2	LSCS
77	N	N	Y	N	N	N	N	N	1	NA	28	37.0	LSCS
78	N	N	Y	N	N	N	N	N	2	NA	26	35.0	LSCS
79	N	N	Y	N	N	N	N	N	1	NA	24	34.6	LSCS
80	N	N	N	Y	N	N	N	N	1	NA	23	33.0	LSCS
81	N	N	Y	N	N	N	N	N	2	NA	28	34.5	LSCS
82	N	N	Y	N	N	N	N	N	1	NA	29	29.0	LSCS
83	N	N	Y	N	N	N	N	N	2	NA	31	35.6	LSCS
84	N	N	Y	N	N	N	N	N	1	NA	28	37.0	LSCS
85	N	N	Y	N	N	N	N	N	2	NA	30	32.4	LSCS
86	N	N	Y	N	N	N	N	N	1	NA	28	28.2	VAGINAL DELIEVERY
87	N	Y	Y	N	N	N	N	N	1	NA	22	41.0	LSCS
88	N	N	Y	N	Y	Y	N	N	1	NA	28	35.0	LSCS
89	N	N	N	N	Y	N	N	N	1	NA	25	37.0	LSCS
90	N	N	N	N	Y	N	N	N	1	NA	24	37.0	LSCS
91	N	N	Y	N	N	N	N	N	1	NA	29	34.3	LSCS
92	N	N	N	N	N	Y	N	N	1	NA	25	34.3	LSCS
93	N	N	Y	N	N	N	N	N	2	NA	25	24.2	VAGINAL DELIEVERY
94	N	N	Y	N	N	N	N	N		NA	26	12.4	VAGINAL EXPULSION AND CHECK CURETTAGE
95	N	N	Y	N	N	N	N	N	1	NA	28	31.2	LSCS
96	N	N	N	N	N	Y	N	N	1	NA	32	38.2	VAGINAL DELIEVERY
97	N	N	N	N	N	Y	N	N	2	NA	28	35.0	LSCS
98	N	N	N	N	N	N	N	N		NA	22	36.0	LSCS
99	N	N	N	N	N	N	N	N		I	27	35.0	LSCS
100	N	N	N	N	N	N	N	N	1	II	26	32.0	LSCS
101	N	N	N	N	N	N	N	N		NA	25	10.0	VAGINAL EXPULSION AND CHECK CURETTAGE
102	N	N	N	N	N	N	N	N		NA	25	8.0	VAGINAL EXPULSION AND CHECK CURETTAGE
103	N	N	N	N	N	N	N	N		I	24	37.0	LSCS
104	N	N	N	N	N	N	N	N		NA	23	20.4	VAGINAL EXPULSION AND CHECK CURETTAGE
105	N	N	N	N	N	N	N	BICUSPID AV- ASYMPTOMATIC		I	25	37.0	LSCS
106	N	N	N	N	N	N	N	N		II	28	20.5	VAGINAL EXPULSION AND CHECK CURETTAGE
107	N	N	N	N	N	N	N	N	1	II	27	33.2	LSCS
108	Y	N	N	N	N	N	N	N	1	II	32	37.0	LSCS
109	N	N	N	N	N	N	N	N		NA	22	38.0	LSCS
110	N	N	N	N	N	N	N	N		I	23	7.0	VAGINAL EXPULSION AND CHECK CURETTAGE
111	N	N	N	Y	N	N	Y	RIGHT HEMIPARESIS	2	NA	27	35.0	LSCS

S.No	HYPERTENSION	BRONCHIAL ASTHMA	HYPOTHYROIDISM	HYPERTHYROIDISM	DM	H/O STROKE	K/C/O CHD	K/C/O RHD	HISTORY OF DVT	OTHERS	CARDIAC Surgery before pregnancy	ECG	QRS	Echo	EF (%)
52	N	N	N	N	N	N	N	Y	N		NONE	NSR	90	PROSTHETIC MITRAL, AORTIC AND	40
53	Y	N	N	N	N	N	N	N	N		NONE	NSR	90	EF 60%, MODERATE CLEAR PERICARDIAL	60
54	N	N	N	N	N	N	N	N	N		NONE	NSR	100	NORMAL LVEF	60
55	Y	N	N	N	N	N	N	N	N		NONE	NRS, LVH	110	NORMAL LVEF	60
56	Y	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	90	MILD LVH, EF 60%	60
57	Y	N	N	N	N	N	N	N	N		NONE	NSR, LVH	100	EF 60 %, MILD TR RVSP 34 MM HG	60
58	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	EF 60%	60
59	N	N	Y	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	90	EF 60%	60
60	Y	N	N	N	N	N	N	N	N		NONE	LVH	110	NORMAL LVEF	60
61	Y	N	N	N	N	N	N	N	N		NONE	LVH	90	NORMAL LVEF	60
62	N	N	N	N	N	N	N	N	N		NONE	NSR	92	EF 60%, MILD TR , RVSP 28 MM HG	60
63	N	N	N	N	N	N	N	N	N		NONE	NSR	86	EF 60%	60
64	N	N	N	N	N	N	N	N	N		NONE	NSR	110	EF 60%	60
65	Y	N	N	N	N	N	N	N	N		NONE	LVH	98	EF 60%	60
66	N	N	Y	N	N	N	N	N	N		NONE	LVH	110	EF 60%	60
67	N	N	N	N	N	N	N	N	N		NONE	NSR	100	EF 60%	60
68	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	EF 60%	60
69	N	N	N	N	Y	N	N	N	N		NONE	NSR	80	EF 60%	60
70	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	98	EF 60%	60
71	Y	N	N	N	N	N	N	N	N		NONE	LVH	110	EF 60%	60
72	N	N	N	N	N	N	N	N	N		NONE	NSR	90	EF 60%	60
73	N	N	N	N	N	N	N	N	N		NONE	NSR	84	EF 60%	60
74	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	EF 60&	60
75	N	N	N	N	N	N	N	N	N		NONE	NSR	100	EF 60%	60
76	Y	N	N	N	N	N	N	N	N		NONE	LVH	110	EF 60%	60
77	Y	N	N	N	N	N	N	N	N		NONE	LVH	90	GROSS LVH , LV MASS 124 GM/M2	60
78	N	N	N	N	N	N	N	N	N		NONE	LVH	100	MOD CONCENTRIC LVH, EF 60%	60
79	N	N	N	N	N	N	N	N	N		NONE	NSR	86	EF 60%	60
80	Y	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	90	EF 60%	60
81	N	N	N	N	N	N	N	N	N		NONE	NSR	100	EF 60%	60
82	N	N	Y	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	98	EF 60%	60
83	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	100	EF 60%	60
84	Y	N	N	N	N	N	N	N	N		NONE	LVH	90	GROSS LVH , LV MASS 135 GM/M2	60
85	Y	N	Y	N	N	N	N	N	N		NONE	LVH	110	EF 60%	60
86	N	N	N	N	N	N	N	N	N		NONE	SINUS RHYTHM	92	EF 60%	60
87	N	N	N	N	N	N	N	N	N		NONE	NSR	90	NORMAL LVEF	60
88	N	N	N	N	Y	N	N	N	N		NONE	LVH	90	NORMAL LVEF	60
89	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	90	NORMAL LVEF	60
90	Y	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	100	NORMAL LVEF	60
91	Y	N	N	N	N	N	N	N	N		NONE	LVH	100	EF 60%	60
92	N	N	N	N	N	N	N	N	N		NONE	NSR	90	NORMAL LVEF	60
93	Y	N	N	N	N	N	N	N	N		NONE	NSR	80	NORMAL LVEF	60
94	Y	N	N	N	N	N	N	N	N		NONE	NSR	86	NORMAL LVEF	60
95	Y	N	N	N	N	N	N	N	N		NONE	NSR	90	NORMAL LVEF	60
96	N	N	N	N	Y	N	N	N	N		NONE	NSR	100	NORMAL LVEF	60
97	N	N	Y	N	N	N	N	N	N		NONE	NSR	80	NORMAL LVEF	60
98	N	N	N	N	N	N	Y	N	N		VSD CLOSURE	NSR	100	TINY RESIDUAL VSD AT DISTAL PART OF	60
99	N	N	N	N	N	N	Y	N	N		NONE	NSR, T WAVE INVERSION IN V1-	120	SEVERE TR, EF 60%	60
100	N	N	N	N	N	N	Y	N	N		NONE	NSR		LARGE PDA WITH LEFT TO RIGHT SHUNT;	48
101	N	N	N	N	N	N	Y	N	N		TOF REPAIR	NSR		VSD PATCH INTACT , NO RVOTO, FREE PR	26
102	N	N	N	N	N	N	Y	N	N		VSD CLOSURE	NSR		RESIDUAL VSD 4 MM PG 90 MM HG LEFT	60
103	N	N	N	N	N	Y	Y	N	N		NONE	SINUS TACHYCARDIA		NORMAL LVEF , NORMAL RA/ RV	60
104	N	N	N	N	N	N	Y	N	N		NONE	SINUS TACHYCARDIA, RBBB	128	RA/RV DILATED , RVSP 48 MM HG , MOD	60
105	N	N	N	N	N	N	N	N	N		NONE	NSR. LAD, LVH	110	SEVERE AS , MOD AR,	60
106	N	N	N	N	N	N	Y	N	N		NONE	NSR	120	SMALL VSD, PG 60 MM HG; RA/RV	60
107	N	N	N	N	N	N	N	Y	N		POST MVR	NSR		PROSTHETIC VALVE NORMAL	60
108	N	N	Y	N	N	N	N	N	N		NONE	ATRIAL FIBRILLATION	120	SEVERE MS WITH SEVERE MR,MVA 0.8	60
109	N	N	N	N	N	N	N	Y	N		NONE	NSR, LAE	90	MOD MS, MOD MR, MVA 1.2 CM2, LA	60
110	N	N	N	N	N	N	N	Y	N		POST MVR	NSR	94	NORMALLY FUNCTIONING PROSTHETIC	62
111	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA		VEGETATION ON AML AND PML	60

S.No	RV DISFUNCTION IF YES RVFAC	RVFAC	PASP	LVH	LVEDD	LVESD	DIASTOLIC DYSFUNCTION	DIASTOLIC DYSFUNCTION TYPE	HEMOGLOBIN	BNP	Congenital HD	Acquired HD	GDMA2	PIH	GESTATIONAL HTN	PRE ECLAMPSIA
52	NO	35	35	YES,MILD	58	39	NO		11.5	150	NONE	PPCM	N	N	N	N
53	NO	35	20	NO	46	26	NO		12		NONE	NONE	N	Y	N	Y
54	NO	35	25	NO	52	30	YES	TYPE 2	10	400	NONE	HFPEF	N	Y	N	N
55	NO	35	25	NO	49	32	YES	TYPE 2	12	150	NONE	HFPEF	N	Y	N	N
56	NO	35	20	NO	35	25	NO		9.2		NONE	NONE	N	Y	Y	N
57	NO	35	25	NO	44	32	YES	TYPE 2	11.5	175	NONE	HFPEF	N	Y	N	N
58	NO	35	28	NO	45	32	NO		9.5		NONE	NONE	N	Y	N	Y
59	NO	35	30	NO	48	32	YES	TYPE 2	12.7	230	NONE	HFPEF	N	Y	N	Y
60	NO	35	20	NO	46	26	YES	TYPE 2	11.5	350	NONE	HFPEF	N	Y	N	N
61	NO	35	24	NO	46	31	NO		13		NONE	NONE	N	Y	N	N
62	NO	35	20	NO	48	38	NO		13.3		NONE	NONE	N	Y	Y	N
63	NO	35	20	NO	43	36	NO		11		NONE	NONE	N	Y	N	N
64	NO	35	25	NO	44	32	NO		12.2		NONE	NONE	N	Y	N	N
65	NO	35	35	NO	45	31	YES	TYPE 1	11.7	5	NONE	NONE	N	Y	N	N
66	NO	35	25	NO	46	30	NO		8.5		NONE	NONE	N	Y	N	N
67	NO	35	20	NO	45	31	NO		12.5		NONE	NONE	N	Y	Y	N
68	NO	35	28	NO	48	24	NO		11.6		NONE	NONE	N	Y	N	N
69	NO	35	35	NO	44	32	YES	TYPE 1	10.5	135	NONE	HFPEF	N	Y	N	N
70	NO	35	30	NO	49	34	YES	TYPE 2	11.4	265	NONE	HFPEF	N	Y	Y	N
71	NO	35	20	NO	48	28	NO		12.6		NONE	NONE	N	Y	N	N
72	NO	35	25	NO	45	23	NO		11.5		NONE	NONE	N	Y	N	N
73	NO	35	30	NO	47	30	NO		12.6		NONE	NONE	N	Y	Y	N
74	NO	35	25	NO	45	32	YES	TYPE 2	10.5	195	NONE	HFPEF	N	Y	N	N
75	NO	35	25	NO	44	32	NO		12.5		NONE	NONE	N	Y	N	Y
76	NO	35	20	NO	40	27	YES	TYPE 2	12		NONE	NONE	N	Y	N	Y
77	NO	35	40	YES,GROSS	40	26	YES	TYPE 2	13		NONE	NONE	N	Y	N	Y
78	NO	35	35	YES,MODERATE	43	27	YES	TYPE 2	11		NONE	NONE	N	Y	Y	N
79	NO	35	20	NO	44	25	NO		12		NONE	NONE	N	Y	N	N
80	NO	35	25	NO	45	31	NO		10.5		NONE	NONE	Y	N	N	N
81	NO	35	20	NO	48	32	NO		12		NONE	NONE	N	Y	N	N
82	NO	35	25	NO	45	26	NO		11		NONE	NONE	N	Y	N	N
83	NO	35	35	NO	48	32	NO		12		NONE	NONE	N	Y	N	N
84	NO	35	35	YES,GROSS	40	26	YES	TYPE 3	13		NONE	NONE	N	Y	N	Y
85	NO	35	20	NO	45	31	YES	TYPE 1	13.5	275	NONE	HFPEF	N	Y	N	N
86	NO	35	20	NO	45	32	NO		10.5		NONE	NONE	N	Y	N	Y
87	NO	35	25	NO	44	29	NO		10.9		NONE	NONE	N	Y	N	N
88	NO	35	28	YES,MODERATE	45	26	YES	TYPE 2	6.5	600	NONE	HFPEF	N	Y	N	N
89	NO	35	30	NO	45	28	NO		8		NONE	NONE	N	Y	N	Y
90	NO	35	20	NO	43	23	NO		7		NONE	NONE	N	Y	N	Y
91	NO	35	28	YES,MILD	49	34	YES	TYPE 2	12.2		NONE	NONE	N	Y	N	N
92	NO	35	25	NO	42	25	NO		11.8		NONE	NONE	Y	N	N	N
93	NO	35	20	NO	42	22	NO		12.5		NONE	NONE	N	Y	N	Y
94	NO	35	20	NO	45	26	NO		11.6		NONE	NONE	Y	N	N	N
95	NO	35	25	YES,MILD	45	25	NO		13.2		NONE	NONE	N	Y	N	Y
96	NO	35		NO			NO				NONE	NONE	N	N	N	N
97	NO	35	20	NO	45	29	YES	TYPE 1	11		NONE	NONE	Y	N	N	N
98	NO	35	35	NO			NO				VSD	NONE	N	N	N	N
99	YES	20	40	NO	43	27	NO		12		EBSTEIN ANAMOLY	NONE	N	N	N	N
100	NO	35	30	NO			NO				PDA	NONE	N	N	N	N
101	YES	26	45	NO	39	24	NO		10		TOF	NONE	N	N	N	N
102	NO	35		NO	47	31	NO				VSD	NONE	N	N	N	N
103	NO	35		NO	46	27	NO				PFO	NONE	N	N	N	N
104	NO	35		NO	46	31	NO		12.5		TOF WITH DORV (UNREPAIRED)	NONE	N	N	N	N
105	NO	35	35	YES,MODERATE	46	26	YES	TYPE 2	9.6		BICUSPID AV	NONE	N	N	N	N
106	NO	35		NO	47	27	NO		10.5		VSD		N	N	N	N
107	NO	35	25	NO	43	30	NO		11.5		NONE	RHD	N	N	N	N
108	NO	35	45	NO	45	32	NO		10		NONE	RHD	N	N	N	N
109	NO	35	55	NO	53	36	NO		11		NONE	RHD	N	N	N	N
110	NO	35	30	NO	43	28	NO		10.5		NONE	RHD	N	N	N	N
111	NO	35	50	NO	52	37	YES	TYPE 2	8.9		NONE	INFECTIVE ENDOCARDITIS	N	N	N	N

S.No	PRE ECLAMPSIA WITH SEVERE FEATURES	ECLAMPSIA	SEPSIS	AKI	VTE	OTHERS	FGR	MACROSMIA	ANHYDROAMNIOS	OLIGOHYDROAMNIOS	POLYHYDRAMNIOS	PPH	PRE TERM	PPROM	FETAL/ PERINATAL COMPLICATIONS	LOW BIRTH WEIGHT (GM)
52	N	N	N	N	N	N	N	N	N	N	N	N	N	N	LOW BIRTH WEIGHT	1400
53	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1000
54	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1200
55	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
56	N	N	N	N	N	N	N	N	N	N	N	N	N	N	LOW BIRTH WEIGHT	1560
57	Y	N	N	N	N	N	Y	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1800
58	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	IUD	
59	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	A-NORMAL,B-1720
60	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1760
61	Y	N	N	N	N	N	Y	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1500
62	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
63	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	A-1980,B-1940
64	Y	N	N	N	N	N	Y	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	A-1780,B-1340
65	Y	N	N	N	N	N	Y	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1320
66	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	IUD	
67	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
68	Y	N	N	N	N	N	Y	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1620
69	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	LOW BIRTH WEIGHT	1360
70	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	LOW BIRTH WEIGHT	A-1920,B-840
71	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1630
72	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	A-1900,B-1960
73	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
74	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
75	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
76	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
77	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
78	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
79	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
80	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	NONE	
81	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	A-1600,B-1860
82	Y	N	N	N	N	N	Y	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1300
83	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
84	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
85	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
86	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	IUD	
87	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
88	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	NONE	
89	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	NONE	
90	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	NONE	
91	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	LOW BIRTH WEIGHT	1900
92	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	NONE	
93	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	IUD	
94	N	N	N	N	N	N	N	N	N	N	N	N	N	N	IUD	
95	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	LOW BIRTH WEIGHT	1700
96	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	IUD	
97	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
98	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
99	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1500
100	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
101	N	N	N	N	N	N	N	N	N	N	N	N	N	N	MISSED ABORTION	
102	N	N	N	N	N	N	N	N	N	N	N	N	N	N	MISSED ABORTION	
103	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
104	N	N	N	N	N	N	N	N	N	N	N	N	N	N	MISSED ABORTION	
105	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
106	N	N	N	N	N	N	N	N	N	N	N	N	N	N	MISSED ABORTION	
107	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
108	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
109	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	NONE	
110	N	N	N	N	N	N	N	N	N	N	N	N	N	N	MISSED ABORTION	
111	N	N	N	Y	N	EMBOLIC STROKE	N	N	Y	N	N	N	N	N	NONE	

S.No	PRIMARY OUTCOMES	MECHANICAL VENTILATOR	BETA BLOCKERS	RAAS #	LOOP DIURETICS	MRA	ANTICOAGULATION	OTHERS	HOSPITAL STAY (DAYS)	CARPREG II SCORING	6mn FOLLOW UP NYHA	EF AT 6 MONTHS	RECOVERED	MORATILTY
52	LEFT VENTRICULAR FAILURE	Y	N	N	N	N	VKA	N	9	3	I	40	NO	N
53	NONE	N	N	Y,ARB	N	N	N	N	6	0	I			N
54	NONE	N	N	N	N	Y	N	SGLT2#	5	0	I	60		N
55	NONE	N	N	Y,ARB	N	N	N	N	5	0	I			N
56	NONE	N	Y	Y,ARB	N	N	N	N	9	0	I	60		N
57	NONE	N	Y	Y,ARB	N	N	N	N	10	0	II	60		N
58	NONE	N	N	Y,ARB	N	N	N	N	4	0	I	60		N
59	NONE	N	Y	N	N	N	N	N	5	0	I	60		N
60	NONE	N	N	Y,ARB	N	N	N	N	6	0	I	60		N
61	NONE	N	N	Y,ARB	N	N	N	N	4	0	I	60		N
62	NONE	N	Y	N	N	N	N	N	12	0	I	60		N
63	NONE	N	N	Y,ARB	N	N	N	N	7	0	I	60		N
64	NONE	N	Y	Y,ARB	N	N	N	N	8	0	I	60		N
65	NONE	N	N	Y,ARB	N	N	N	N	6	0	I	60		N
66	NONE	N	Y	N	N	N	N	N	3	0	I	60		N
67	NONE	N	N	Y,ARB	N	N	N	N	3	0	I	60		N
68	NONE	N	Y	N	N	N	N	N	6	0	I	60		N
69	NONE	N	Y	Y,ARB	N	Y	N	N	4	0	I	60		N
70	NONE	N	N	Y,ARB	N	Y	N	N	6	0	I	60		N
71	NONE	N	Y	N	N	N	N	N	6	0	I	60		N
72	NONE	N	Y	Y,ARB	N	N	N	N	7	0	I	60		N
73	NONE	N	Y	Y,ARB	N	N	N	N		0	I			N
74	NONE	N	Y	N	N	Y	N	N	6	0	I	60		N
75	NONE	N	N	Y,ARB	N	N	N	N		0	I	60		N
76	NONE	N	Y	N	N	N	N	N		0	I	60		N
77	NONE	N	Y	Y,ARB	N	N	N	N		0	I	60		N
78	NONE	N	N	N	N	N	N	CCB		0	I			N
79	NONE	N	Y	N	N	N	N	N	9	0	I	60		N
80	NONE	N	N	Y,ARB	N	N	N	INSULIN	7	0	I	60		N
81	NONE	N	Y	N	N	N	N	N		0	I	60		N
82	NONE	N	N	Y,ARB	N	N	N	N	3	0	I	60		N
83	NONE	N	N	Y,ARB	N	N	N	N	7	0	I	60		N
84	NONE	N	Y	Y,ARB	N	N	N	N		0	I	60		N
85	NONE	N	Y	Y,ARB	N	N	N	N	7	0	I	60		N
86	NONE	N	Y	N	N	N	N	N	3	0	I			N
87	NONE	Y	Y	Y,ARB	N	N	N	N	8	4	I	60		N
88	MATERNAL DEATH	N	N	N	N	N	N	N	3	0	MATERNAL DEATH			Y
89	NONE	N	N	N	N	N	N	N	2	0	I	60		N
90	NONE	N	N	N	N	N	N	N	2	0	I	60		N
91	NONE	N	Y	N	N	N	N	CCB	6	0	I			N
92	NONE	N	N	N	N	N	N	INSULIN	4	0	I			N
93	NONE	N	N	N	N	N	N	CCB	5	0	I			N
94	NONE	N	Y	N	N	N	N	N	3	0	I			N
95	NONE	N	N	N	N	N	N	CCB	5	0	I			N
96	NONE	N	N	N	N	N	N	N		0	I			N
97	NONE	N	N	N	N	N	N	N		0	I			N
98	NONE	N	N	N	N	N	N	N	6	0	I			N
99	NONE	N	N	N	N	N	N	N	5	1	I	60		N
100	NONE	N	N	N	N	N	N	N		1	I			N
101	NONE	N	N	N	N	N	N	N	3	0	II			N
102	NONE	N	N	N	N	N	N	N		0	I			N
103	NONE	N	N	N	N	N	N	N	5	0	I			N
104	NONE	N	N	N	N	N	N	N	3	0	I			N
105	NONE	N	N	N	N	N	N	N	6	2	I	60		N
106	NONE	N	N	N	N	N	N	N	4	2	II			N
107	NONE	N	N	N	N	N	N	N		3	I			N
108	ACUTE PULMONARY EDEMA	N	N	N	Y	Y	VKA	VERAPAMIL	7	5	II	60		N
109	NONE	N	Y	N	N	Y	VKA	N	7	2	I	60		N
110	NONE	N	N	N	N	N	VKA	DILTIAZEM	3	3	I	60		N
111	ACUTE PULMONARY EDEMA	Y	N	N	Y	Y	N	IVABRADINE	30	8	II			N

S.No	Name	DIAGNOSIS	MRD NUMBER	D.O.A/ OPD DATE	Age at Conception	BOOKED/ UNBOOKED	ADMITTED AT HEART CENTRE	PRE LSCS/ VD	POST LSCS/ VD	POST PARTUM (DAYS)	POST PARTUM (DAYS) GP	Parity 1	ABOORTION	TWINNPREGNANCYES	ivf treatment	SHORTNESS OF BREATH
112	RK	VHD	1532773	OPD 12-02-2023	34	UNBOOKED	NO	YES	NO		N	G2A1	Y	NO	NO	N
113	AG	VHD	1476372	21-09-2023	31	BOOKED	NO	NO	NO		N	PRIMI	N	NO	NO	Y
114	AT	VHD	1542165	08-10-2023	42	UNBOOKED	NO	NO	NO		N	G2P1L1	N	NO	NO	N
115	CK	VHD	1439696	25-03-2024	25	BOOKED	NO	NO	NO		N	PRIMI	N	NO	NO	N
116	PS	VHD	481162	OPD 19-11-2023	37	UNBOOKED	NO	YES	NO		N	G3P2L1	N	NO	NO	N
117	SK	VHD	1614499	OPD 27-10-2023	32	UNBOOKED	YES	YES	NO		N	G3P1L1	N	NO	NO	Y
118	KR	VHD	1339400	05-04-2024	31	UNBOOKED	NO	NO	NO		N	G2P1L1	N	NO	YES	N
119	KK	VHD	1483047	14-02-2024	28	UNBOOKED	NO	NO	NO		N	PRIMI	N	NO	NO	N
120	GK	VHD	1768912	11-06-2024	28	UNBOOKED	NO	NO	NO		N	G2P1L1	N	NO	NO	N
121	SK	VHD	1202578	OPD 12-07-2023	34	BOOKED	NO	YES	NO		N	G5P4L1	N	NO	NO	N
122	SB	VHD	1357789	OPD 10-08-2023	38	UNBOOKED	NO	YES	NO		N	G3P2L2	N	NO	NO	N
123	AT	VTE	1555804	OPD 21-07-2023	29	UNBOOKED	YES	YES	NO		N	PRIMI	N	NO	NO	N
124	PA	VTE	1303459	OPD 17-01-2024	33	UNBOOKED	YES	YES	NO		N	G2P1L1	N	NO	NO	N
125	MK	VTE	362452	12-07-2023	27	BOOKED	YES	NO	NO		N	G2P1L1	N	NO	NO	N
126	HKS	VTE	213934	31-12-2022	48	BOOKED	NO	YES	NO		N	G2A1	Y	NO	YES	N
127	DG	VTE	1022684	15-06-2023	27	UNBOOKED	YES	NO	NO	4	Y	P2L2	N	NO	NO	Y
128	SK	VTE	1556734	21-07-2023	32	UNBOOKED	YES	NO	YES	40	Y	PRIMI	N	NO	NO	Y
129	RG	OTHER	1572918	05-03-2024	36	BOOKED	NO	NO	NO		N	G3P2L2	N	NO	NO	N
130	MK	OTHER	1556725	04-08-2023	26	UNBOOKED	NO	NO	NO		N	G2P1A1	Y	NO	NO	Y
131	JK	OTHER	1600088	02-10-2023	32	UNBOOKED	YES	YES	NO		N	PRIMI	N	NO	NO	Y
132	SS	OTHER	1622075	11-11-2023	26	UNBOOKED	YES	NO	YES	1	Y	P1A1	Y	NO	NO	Y
133	HK	OTHER	1364600	OPD 26-01-2023	25	BOOKED	NO	YES	NO		N	PRIMI	N	NO	NO	N
134	MK	OTHER	1501332	18-04-2023	22	UNBOOKED	YES	NO	YES		N	PRIMI	N	NO	NO	N
135	M	OTHER	1471311	27-02-2023	32	UNBOOKED	NO	NO	NO		N	G2P1L1	N	NO	NO	Y

S.No	PALPITATIONS	GTCS	INCREASED BP READINGS	FEVER	POST PARTUM HEMORRHAGE	INCREASED SUGARS	SHOCK	OTHERS	DURATION OF SYMPTOMS (DAYS) GP	NYHA CLASS (ADMSN)	BMI	PREGNANCY DURATION (TERMINATION)	mode of delievery
112	N	N	N	N	N	N	N	N		NA	20	34.0	LSCS
113	Y	N	N	N	N	N	N	N	1	II	24	35.1	LSCS
114	N	N	N	N	N	N	N	N		NA	26	35.5	LSCS
115	Y	N	N	N	N	N	N	N	1	II	21	37.2	VAGINAL DELIEVERY
116	N	N	N	N	N	N	N	N		NA	24	35.5	LSCS
117	N	N	N	N	N	N	N	N	1	II	21	34.5	LSCS
118	N	N	N	N	N	N	N	N		I	22	37.0	VAGINAL DELIEVERY
119	N	N	N	N	N	N	N	N		I	26	38.0	VAGINAL DELIEVERY
120	N	N	N	N	N	N	N	N		NA	23	19.1	VAGINAL EXPULSION AND CHECK CURETTAGE
121	N	N	N	N	N	N	N	N		NA	25	32.0	LSCS
122	N	N	N	N	N	N	N	N		NA	20	32.0	LSCS
123	N	N	N	N	N	N	N	N		NA	24	37.2	LSCS
124	N	N	N	N	N	N	N	N		NA	42	35.0	VAGINAL DELIEVERY
125	N	N	N	N	N	N	N	N		NA	24	38.0	LSCS
126	N	N	N	N	N	N	N	N		NA	29	35.2	LSCS
127	Y	N	N	N	N	N	N	N	1	IV	25	37.0	LSCS
128	N	N	N	N	N	N	N	LOWER LIMB SWELLING	1	III	27	35.0	VAGINAL DELIEVERY
129	Y	N	N	N	N	N	N	N	1	II	28	37.0	VAGINAL DELIEVERY
130	N	N	N	N	N	N	N	N	2	III	26	35.0	LSCS
131	N	N	N	N	N	N	N	CHEST PAIN	1	III	27	34.1	LSCS
132	N	N	N	Y	N	N	Y	N	1	II	26	34.5	LSCS
133	N	N	N	N	N	N	N	H/0 MYOCARDITIS		I	22	37.0	VAGINAL DELIEVERY
134	N	N	N	Y	N	N	N	N	1	NA	20	38.0	LSCS
135	N	N	N	N	N	N	N	N	1	IV	26	9.6	VAGINAL EXPULSION AND CHECK CURETTAGE

S.No	HYPERTENSION	BRONCHIAL ASTHMA	HYPOTHYROIDISM	HYPERTHYROIDISM	DM	H/O STROKE	K/C/O CHD	K/C/O RHD	HISTORY OF DVT	OTHERS	CARDIAC Surgery before pregnancy	ECG	QRS	Echo	EF (%)
112	N	N	N	N	N	N	N	Y	N		NONE	NSR		MILD MS, MODERATE MR	60
113	N	N	N	N	N	N	N	N	N		NONE	ATRIAL FIBRILLATION	110	MODERTAE MS	60
114	N	N	N	N	N	N	N	Y	N		NONE	NSR	90	MODERATE MR	60
115	N	N	N	N	N	N	N	N	N		NONE	NSR	90	MILD MS	60
116	N	N	N	N	N	N	N	Y	N		NONE	NSR	100	MILD MS, MILD MR, MODERATE AR	60
117	N	N	N	N	N	N	N	Y	N		NONE	NSR	90	MODERATE MS, MVA 1.7 CM2	60
118	Y	N	N	N	N	N	N	N	N		NONE	NSR	90	MILD MR ; MVA 3.2 CM2;	60
119	N	N	N	N	N	N	N	N	N		NONE	NSR	80	MILD MS, MVA 2.2 CM2	60
120	N	N	N	N	N	N	N	Y	N		NONE	ATRIAL FIBRILLATION	110	MODERATE MS, MVA -1.5 CM2	60
121	N	N	N	N	N	N	N	N	N		NONE	NSR		SEVERE MS, MVA 0.8 CM2;	60
122	N	N	N	N	N	N	N	Y	N		POST MVR	NSR		PROSTHETIC MITRAL VALVE NORMAL	60
123	N	N	N	N	N	N	N	N	Y		NONE	NSR		NORMAL CARDIAC CHAMBERS	60
124	N	N	N	N	N	N	N	N	Y		NONE	SINUS TACHYCARDIA	90	NORMAL LVEF, RA/RV NORMAL	60
125	N	N	N	N	N	N	N	N	Y		NONE	NSR	80	NORMAL LVEF , NORMAL RA/ RV	60
126	N	N	Y	N	N	N	N	N	Y		NONE	SINUS TACHYCARDIA	100	NORMAL LVEF, NORMAL RA/RV	60
127	N	N	N	N	N	N	N	N	Y		NONE	SINUS TACHYCARDIA	110	RA/RV MODERATELY DILATED;	60
128	N	N	N	N	N	N	N	N	Y		NONE	NSR, RBBB	110	RA/RV MILDLY DILATED	60
129	Y	N	N	N	N	N	N	N	N		NONE	LVH	110	HYPERTROPHIC CMP(MID VARIANT), NO	60
130	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	110	RA RV MOD DILATED	60
131	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA, RBBB	110	RA/RV GROSSLY DILATED	60
132	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	100	NORMAL LVEF	60
133	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	84	NORMAL LVEF	60
134	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA	92	MILD GHK OF LV; EF 42% ;	42
135	N	N	N	N	N	N	N	N	N		NONE	SINUS TACHYCARDIA, IVCD	136	GHK OF LV ,LA/ LV DILATED	22

S.No	RV DISFUNCTION IF YES RVFAC	RVFAC	PASP	LVH	LVEDD	LVESD	DIASTOLIC DYSFUNCTION	DIASTOLIC DYSFUNCTION TYPE	HEMOGLOBIN	BNP	Congenital HD	Acquired HD	GDMA2	PIH	GESTATIONAL HTN	PRE ECLAMPSIA
112	NO	35	30	NO			NO		9.5		NONE	RHD	N	N	N	N
113	NO	35	35	NO	42	24	NO		11.2		NONE	RHD	N	N	N	N
114	NO	35	25	NO			NO		12.5		NONE	RHD	N	N	N	N
115	NO	35	25	NO	45	32	NO		10		NONE	RHD	N	N	N	N
116	NO	35	20	NO	52	39	NO		13		NONE	RHD	N	N	N	N
117	NO	35	25	NO	42	27	NO		10		NONE	RHD	N	N	N	N
118	NO	35	25	NO	44	28	NO		12		NONE	RHD	N	N	N	N
119	NO	35	20	NO	47	31	NO		11		NONE	RHD	N	N	N	N
120	NO	35	30	NO	45	26	NO		12.3		NONE	RHD	N	N	N	N
121	NO	35	40	NO	46	24	NO		10		NONE	RHD	N	N	N	N
122	NO	35	35	NO	41	23	NO		10.5		NONE	RHD	N	N	N	N
123	NO	35	20	NO	46	21	NO		12.3		NONE	NONE	N	N	N	N
124	NO	35	25	NO	45	24	NO		11	100	NONE	NONE	N	Y	Y	N
125	NO	35	22	NO	46	26	NO		12		NONE	NONE	N	N	N	N
126	NO	35	30	NO	44	23	NO		12		NONE	NONE	N	N	N	N
127	YES	22	60	NO	43	26	NO		12.1		NONE	RV DYSFUNCTION	N	N	N	N
128	YES	28	40	NO	45	31	NO		8.6		NONE	RV DYSFUNCTION	N	N	N	N
129	NO	35	25	YES,MODERATE	44	24	YES	TYPE 2	11.4		NONE	HCM	N	N	N	N
130	YES	26	50	NO	46	26	NO		12		NONE	PAH	N	N	N	N
131	YES	22	60	NO	36	24	NO		12		NONE	PAH	N	N	N	N
132	NO	35	35	NO	44	24	NO		8.8		NONE	MYOCARDITIS	N	N	N	N
133	NO	35	20	NO	46	26	NO		12.2	125	NONE	H/O MYOCARDITIS	N	N	N	N
134	NO	35	20	NO	47	37	YES	TYPE 3	11.6	5	NONE	MYOCARDITIS	N	Y	N	Y
135	NO	35	25	NO	64	42	YES	TYPE 3	10.4	300	NONE	DCMP	N	N	N	N

S.No	PRE ECLAMPSIA WITH SEVERE FEATURES	ECLAMPSIA	SEPSIS	AKI	VTE	OTHERS	FGR	MACROSMIA	ANHYDROAMNIOS	OLIGOHYDROAMNIOS	POLYHYDRAMNIOS	PPH	PRE TERM	PPROM	FETAL/ PERINATAL COMPLICATIONS	LOW BIRTH WEIGHT (GM)
112	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
113	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
114	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
115	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
116	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
117	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1800
118	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
119	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
120	N	N	N	N	N	N	N	N	N	N	N	N	N	N	MISSED ABORTION	
121	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1600
122	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	LOW BIRTH WEIGHT	1300
123	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	NONE	
124	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	NONE	
125	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	NONE	
126	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	NONE	
127	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	NONE	
128	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	IUD	
129	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
130	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
131	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	NONE	
132	N	N	Y	N	N	N	N	N	N	N	N	Y	Y	N	IUD	
133	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
134	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	
135	N	N	N	N	N	N	N	N	N	N	N	N	N	N	NONE	

S.No	PRIMARY OUTCOMES	MECHANICAL VENTILATOR	BETA BLOCKERS	RAAS #	LOOP DIURETICS	MRA	ANTICOAGULATION	OTHERS	HOSPITAL STAY (DAYS)	CARPREG II SCORING	6mn FOLLOW UP NYHA	EF AT 6 MONTHS	RECOVERED	MORATILTY
112	NONE	N	Y	N	N	N	N	N		1	I			N
113	NONE	N	Y	N	N	N	VKA	N	5	2	I			N
114	NONE	N	N	N	N	N	N	N	4	1	I			N
115	NONE	N	N	N	N	N	N	N	4	0	I	60		N
116	NONE	N	Y	Y,ARB'S	N	N	N	N	6	1	I	60		N
117	NONE	N	Y	N	N	N	VKA	N	6	0	II	60		N
118	NONE	N	N	N	N	N	N	N	4	0	I	60		N
119	NONE	N	N	N	N	N	N	N	4	0	I	60		N
120	NONE	N	N	N	N	N	N	N	3	2	I			N
121	NONE	N	N	N	N	N	N	N		2	II			N
122	NONE	N	N	N	N	N	VKA	VERAPAMIL		3	I			N
123	NONE	N	N	N	N	N	N	N		0	I			N
124	NONE	N	N	N	N	N	VKA	N	4	2	I	60		N
125	NONE	N	N	N	N	N	VKA	N	5	0	I	60		N
126	NONE	N	N	N	N	N	VKA	N	7	1	I	60		N
127	RIGHT HEART FAILURE	N	N	N	N	Y	NOAC	N	14	4	I	60		N
128	RIGHT HEART FAILURE	N	N	N	N	N	VKA	N	8	4	I	60		N
129	NONE	N	Y	N	N	N	N	N	4	2	I	60		N
130	NONE	N	N	N	N	Y	N	N	5	4	II	60		N
131	NONE	N	N	N	N	N	N	SILDENAFIL	4	2	II	60		N
132	NONE	N	N	N	N	N	N	N	8	4	I	60		N
133	NONE	N	N	N	N	N	N	N	4	2	I	60	YES	N
134	NONE	N	N	Y,ACE-I	N	N	N	STATINS	4	2	II	45	NO	N
135	LEFT VENTRICULAR FAILURE	N	Y	N	Y	Y	N	N	5	5	II	40	NO	N

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The Principal
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Subject:: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 25.09.2023 has approved the plan of thesis of **Sahil Bansal**, a student of your college for **DM Cardiology** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Prevalence, clinical profile, risk factors & materno-fetal outcomes in pregnant females with cardiovascular ailments in a tertiary care centre in north india .

Supervisor	**Co-Supervisor:-**	**Co-Supervisor:-**
Dr. Shibba Takkar Chhabra Professor Dept. of Cardiology	Dr. Ashima Taneja Prof. & Head Dept. of Obstetrics & Gynaecology	Dr. Suman Puri Professor Dept. of Obstetrics & Gynaecology

The student may submit the thesis in triplicate alongwith the plan in triplicate also, to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She may be directed to attach a photocopy of this letter with the forwarding letter while submitting thesis in the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic, if any.

Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTORATE OF MEDICINE

IN

CARDIOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

NOVEMBER, 2022

DR. SAHIL BANSAL

DEPARTMENT OF CARDIOLOGY
DAYANAND MEDICAL COLLEGE & HOSPITAL
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ABSTRACT OF PLAN OF THESIS

Title	PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA
For the degree of	DM CARDIOLOGY
Name of the candidate	Dr. Sahil Bansal
Supervisor	Dr. Shibba Takkar Chhabra, Professor, Department of Cardiology, Dmch Ludhiana.
Co-supervisors	Dr. Ashima Taneja, Professor & Head, Department of Obst. & Gynae, Dmch Ludhiana. Dr. Suman Puri, Professor, Department of Obst. & Gynae, Dmch Ludhiana.
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Many cardiac diseases during pregnancy are under investigation, and many others which are still not understood require further inquiry. Some of these diseases may be exacerbation's of preexisting conditions that the pregnant woman may already have, or they may develop a new disease process that presents because of the complex hormonal changes and physiology of pregnancy. Preexisting conditions that can predispose pregnant women to cardiovascular disease include hypertension, diabetes mellitus, and congenital heart disease.

Aims and objectives: To study the prevalence, mortality/ morbidity data and maternal/fetal outcomes of cardiovascular disease in pregnant women.

Materials & Methods:It will be a single centre, prospective observational study will be conducted on OPD and IPD patients of a tertiary care centre in Ludhiana, Punjab. All pregnant women diagnosed with congenital heart disease, rheumatic heart disease , coronary artery disease, thrombotic disease etc and postpartum females with CVS complications will be included in study.

Clinical Significance: Cardiac ailments are notably increasing in pregnancy as a significant number of women with congenital and valvular heart disease now survive to this age. Also the prevalence of antenatal heart disease are contrasting between high-income, industrialized, and low- and middle-income countries. Most women of reproductive age use their obstetrician-gynecologist as their primary care provider, these physicians are uniquely positioned to identify and refer patients early for management of hypertension, diabetes, and dyslipidemia in order to prevent CVD and stroke. Cardio-obstetrics has emerged as a speciality which allows physician and an obstetrician to work in cohesion during this period.

Keywords: Preeclampsia, cardiovascular disease, maternal health, pregnancy complications.

(Signature)	(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-Supervisor	Co-Supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA"** exist in the Department of Cardiology, DMC&H, and Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate himself under our supervision and guidance.

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APPROVAL PROFORMA
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Name of candidate	Dr. Sahil Bansal
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Topic of Thesis	PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA
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Dr. Harpreet Kaur (Professor & Head, Pathology)	Mr. R K Sharma (Legal Expert)
Dr. Jaspal Singh (Professor & Head, Surgery)	Dr. Ramit Mahajan (Associate Professor, Gastroenterology)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
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Approved: Yes / No

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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

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4	DATE OF ADMISSION OF COURSE	27/04/2022
5	TITLE OF THE TOPIC	PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA

6. BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

In pregnant women with heart disease, the complex physiologic changes of pregnancy may result in maternal and fetal morbidity and mortality, more so if the pathology has compromised the cardiac muscle in subtle or overt ways.

The incidence of pregnancies with cardiovascular disease is rising across India. This is due to the increase in number of women with congenital heart disease(CHD) undergoing partial or complete (surgical or interventional) correction before child bearing age. Even though the overall incidence of rheumatic heart disease(RHD) is decreasing, it remains to be highly prevalent heart disease in our country because of existing cases.

Approximately 1.3% of pregnant women are estimated to have cardiac disease, which can have a significant effect on both maternal and fetal outcomes .[1]

During pregnancy, many changes occur in the cardiovascular system (heart and blood vessels) that increase the stress and workload of the heart. These changes usually start during the first 5-8 weeks of pregnancy and reach their peak in the second trimester (13th to 27th week of pregnancy). During this time, complications related to the heart can develop, especially if women have a pre-existing heart condition.

Cardiovascular Physiology during Pregnancy :

Changes in cardiac function become apparent during the first 8 weeks of pregnancy. Compared with pre-pregnancy measurements, brachial SBP, DBP and central SBP are all significantly lower 6 to 7 weeks from the LMP. The resting pulse rate rises approximately 10beats/min during pregnancy.

HEART : As the diaphragm becomes progressively elevated, the heart is displaced to the left and upward and is rotated on its long axis. As a result, the apex is moved somewhat laterally from its usual position and produces larger cardiac silhouette in chest radio-graphs.

ECG : Slight left-axis deviation due to the altered heart position, Q waves in leads II, III, and avF and flat or inverted T waves in leads III and V1-V3.

CARDIAC SOUNDS :

- Exaggerated splitting of the S1 and increased loudness of both components.
- No definite changes in S2.
- Loud and easily heard S3.
- Systolic murmur is intensified. Ejection systolic murmur >=3/6 and Diastolic murmurs indicate heart disease.
- Continuous murmurs arising from the breast vasculature may be heard.

CARDIAC OUTPUT : CO is increased as early as the 5th week and reflects a reduced SVR and an increased HR. There is an increase of 30-35% in CO. It is maximum - Immediate postpartum> 2nd stage of labour> late 1st stage of labour> 30-32 weeks of pregnancy. There is an increase in coronary blood flow as coronary arteries become sensitive to stress-induced vasodilation. Venous return increases dramatically during pregnancy. ,

BLOOD PRESSURE : Blood pressure (BP) falls up to midterm due to fall in vascular resistance and then BP gradually rises.There is a fall in BP by 5 to 10 mm Hg, with most of the fall during early part of pregnancy. The fall in BP is seen in both SBP and DBP with the fall more prominent in DBP.

ACOG Classification of Hypertension in Pregnancy(2013)[2]

Condition	Definition	Prevalence
GESTATIONAL HYPERTENSION	De novo BP elevations (>140/90 mm Hg) after 20 wks of gestation without other organ system dysfunction	6-7
PRE ECLAMPSIA	De novo BP elevations after 20 wks of gestation coupled with proteinuria or other end-organ dysfunction	5-7
CHRONIC HYPERTENSION	Elevated BP before 20 wks of gestation or persisting beyond 12 wks postpartum	1-5
CHRONIC HYPERTSNION WITH SUPERIMPOSED PRE ECLAMPSIA	Increased BP and new-onset proteinuria or other end-organ dysfunction in addition to preexisting hypertension	0.2-1

HEART RATE : Heart rate increase progressively till the end of pregnancy reaching its peak in the 3rd trimester. About 25% change in HR from the baseline values has been noted. Increase in sympathetic activity as compensatory mechanism to peripheral vasodilation during pregnancy explains the increase in HR. Increased secretion of estradiol-stimulating nitric oxide synthesis leading to β-adrenergic–mediated vasodilation.

HYPERCOAGUBILITY: VTE is approximately 10-times more common in the pregnant population (compared with non-pregnant women) with an incidence of 1 in 1000 and the highest risk in the postnatal period.[3]

A summary of the procoagulant changes that occur in the blood system during pregnancy:

- Factors II, V IX and protein C- Unchanged.
- Increased Concentration of factors VII, VIII, X and vWF and pronounced increases in fibrinogen

- Decreased Protein S
- Plasminogen activator inhibitor type 1 levels are increased five-fold
- PAI-2 produced by the placenta increaseed dramatically during third trimester

Management of cardiac disease in a pregnant woman should ideally start before or during the early gestation. Signs and symptoms of pregnancy can mimic heart disease. For women with pre-existing heart disease, the most common cardiac complications during pregnancy are arrhythmia's, heart failure (HF), and thromboembolic events.

For risk stratification various scores have been formulated from time to time over the last two decades namely, CARPREG I, CARPREG II, ZAHARA, and the WHO score.

The modified World Health Organization (mWHO) classification is widely used.[4]

MODIFIED WORLD HEALTH ORGANIZATION (MWHO)CLASSIFICATION :

Obstetric patients with heart disease are classified into:

Class I: Very low risk

Class II: Low to moderate risk

Class III: High risk

Class IV: Extremely high risk – pregnancy is contraindicated

The multicentre CARPREG (Cardiac Disease in Pregnancy Study) developed a risk index to predict the likelihood of maternal cardiac complications from general maternal, clinical and echocardiographic data, obtained during the baseline antepartum visit.[5]

Subsequently ZAHARA (Zwangerschap bij Aangeboren hartafwijking-II [Pregnancy in Women with Congenital Heart Disease]) was a weighted risk score that included components of the CARPREG risk index.[6]

A significant development was the development of the CAPREG II risk score which seems to provide refinement in decision-making in pregnancy.

CAPREG II was a prediction model incorporating specific cardiac diagnosis, general cardiac variables, and factors related to process of care. Each model has its strengths based on simplicity, applicability and even some complexity when details of specific lesions are factored in.[7]

CARPREG II RISK SCORE :

CARPREG II has been published in May 2018 in the Journal of American College of Cardiology. It improves upon previous CARPREG scoring system and is based on nearly two thousand pregnancies with heart disease from two advanced centres in Canada. The score is based on ten predictors. Each predictor is assigned a weighted point score. The sum of points represents the risk score.

CARPREG II RISK PREDICTORS AND THEIR POINTS:

1. Prior cardiac events or arrhythmias: 3
2. Baseline NYHA class III-IV: 3
3. Mechanical valve: 3
4. Ventricular dysfunction: 2
5. High risk left sided valve disease/LVOT obstruction:2
6. Pulmonary hypertension: 2
7. Coronary artery disease: 2
8. High risk aortopathy: 2
9. No prior cardiac intervention: 1

10. Late pregnancy assessment: 1

Although the scores mentioned above do exist, and may be used to predict mortality, they were never developed within the population in India or other lower-middle income countries (LMIC) and hence were never validated properly, limiting their applicability in a LMIC. There are also unique features that apply to India and other LMIC countries including a lower percentage of corrected lesions, more advanced disease, late presentation during pregnancy, and the different profile of cardiac conditions compared to high income countries (HICs).

6.2 REVIEW OF LITERATURE

In a meta analysis study done in South Asia(2022) to study the prevalence and spectrum of heart disease in pregnant women. After various stages of screening 25 studies were included in the final quantitative synthesis. The pooled prevalence of heart disease among pregnant women was 1.46%.among those with heart disease, 70.25% had rheumatic heart disease and 18.10% had congenital heart disease. The pooled prevalence of preterm labor and delivery among pregnant women with heart disease was 17.63%. Similarly, the pooled maternal and fetal mortality rates were 26.14 and 50.48 per 1000 pregnant women with heart disease respectively.[8]

In a recent retrospective study (2022) conducted in England by Williams et al[9] to compare the risk of cardiovascular disease outcomes for women with and without a history of gestational hypertension and pre-eclampsia using national hospital admissions data.Women who had one or more singleton live births in England between 1997 and 2015 were included in the analysis.During 18 years of follow-up, the number and incidence of total CVD for normotensive women, women with prior gestational hypertension and women with prior pre-eclampsia were 57.1 per 100,000 person-years; 85.8 per 100,000 person-years; and 99.3 per 100,000 person-years, respectively. among cardiovascular disease sub-types, gestational hypertension was strongly associated with dilated cardiomyopathy and unstable angina. Pre-eclampsia was strongly associated with hypertrophic cardiomyopathy and acute myocardial infarction.

In a retrospective study(2019) of 180 patients conducted by Alsheef et al[10] to elucidate the circumstances surrounding pregnancy-induced deep vein thrombosis (DVT) and pulmonary embolism (PE), assess potential factors triggering thrombosis (e.g., thrombophilia, obesity, age, parity, and family history), initial and long-term management, and assess recurrence rate and mortality for VTE in pregnant Saudi women and it was found that 60% and 40% of the VTE cases occurred during

the postpartum and antenatal periods, respectively. Cesarean section was the most prevalent risk factor among study participants ((47.8%)), followed by obesity ((40.6%)). The most common clinical presentations were lower leg pain (57.2%) and lower limb swelling (54.4%). VTE recurrences were observed in approximately 11% of the participants, and maternal mortality occurred in 2 (1.1%) cases. It was Concluded that pregnancy was the most common provoking factor for VTE in our study.

According to a study conducted in AIIMS new delhi in 2018, the total prevalence of congenital heart disease was found to be 9/1000 and this study also indicated that in pregnant women, rheumatic heart disease was the commonest heart condition (78%) of which mitral stenosis was the commonest heart defect (71.54%). Congenital heart disease (18.7%) is the second most common heart condition in pregnancy. This study also showed that those who were operated had fewer complications.[1]

In 2018, Silversides C K et al[7], conducted a study on 1,938 pregnant women, Cardiac complications occurred in 16% of pregnancies and were primarily related to arrhythmias and heart failure. Although the overall rates of cardiac complications during pregnancy did not change over the years, the frequency of pulmonary edema decreased (8% from 1994 to 2001 vs. 4% from 2001 to 2014).

In a study named “Pregnancy outcomes in women with rheumatic mitral valve disease” conducted by Iris M van Hagen et al[11] in 2018 on 390 patients showed that Mitral stenosis (MS) with or without mitral regurgitation (MR) was present in 273 women, isolated MR in 117. The degree of MS was mild in 20.9%, moderate in 39.2%, severe in 19.8%, and severity not classified in the remainder. Maternal death during pregnancy occurred in 1 patient with severe MS. Hospital admission occurred in 23.1% of the women with MS, and the main reason was heart failure (mild MS 15.8%, moderate 23.4%, severe 48.1%; $P<0.001$). Heart failure occurred in 23.1% of patients with moderate or

severe MR. An intervention during pregnancy was performed in 16 patients, 14 had percutaneous balloon mitral commissurotomy, and 2 had surgical valve replacement (1 for MS, 1 for MR). Follow-up at 6 months postpartum was available for 53%, and 3 more patients died (1 with severe MS, 1 with moderate MS, 1 with moderate to severe MR).

In a meta analysis done by steinberg ZL et al[12] (2017) to study maternal and fetal outcomes in pregnant women with mechanical heart valves (MHVs) receiving different methods of anticoagulation.Total 800 pregnancies from 18 publications were included in the analysis. maternal outcomes included death, thromboembolism, and valve failure and fetal outcomes included spontaneous abortion, death, and congenital defects. The studies explored the use of one or more of the following anticoagulants: 1) a vitamin K antagonist (VKA) throughout pregnancy, 2) low molecular weight heparin (LMWH) throughout pregnancy, 3) LMWH for the first trimester followed by VKA (LMWH + VKA), or 4) unfractionated heparin for the first trimester followed by a VKA (UFH + VKA).Composite maternal risk was lowest with VKA (5%) as compared to LMWH (16%), LMWH + VKA (16%), and UFH + VKA (16%). Composite fetal risk was higher with VKA (39%) as compared to LMWH (13%). Similar composite fetal risk was seen with LMWH + VKA (23%) and UFH + VKA (34%). No significant difference in fetal risk was observed between individuals on ≤5 mg of daily warfarin and those on a LMWH regimen.

In 2017 study(meta analysis on 2,58,000 pre eclampsia patients) was conducted by Wu et al[13] to evaluate and quantify the evidence on the relationship between preeclampsia and the future risk of heart failure, coronary heart disease, composite cardiovascular disease, death because of coronary heart or cardiovascular disease, stroke, and stroke death after preeclampsia. It was concluded that preeclampsia is associated with a 4-fold increase in future incident heart failure and a 2-fold increased risk in coronary heart disease, stroke, and death

because of coronary heart or cardiovascular disease after adjusting for age , body mass index and diabetes mellitus.

In a metanalysis done by S De Haas et al[14] in 2017(total 48 studies were included from 1997 to 2016) showed that During normotensive pregnancy, most cardiac geometric indices change from the second trimester onwards. Both Left Ventricular Mass and Relative Wall Thickness increase by 20% and 10%, respectively, consistent with concentric rather than eccentric remodeling while Cardiac adaptation in hypertensive pregnancy deviates from that in healthy pregnancy by a greater change in LVM (95% increase from reference) and RWT (56% increase from reference).

In a study conducted by Mcnamara DM et al[15](2015) to prospectively evaluate recovery of the left ventricular ejection fraction (LVEF) and clinical outcomes in the multicenter IPAC (Investigations of Pregnancy Associated Cardiomyopathy) study.total 100 women with PPCM were included and LVEF was assessed by echocardiography at baseline and at 2, 6, and 12 months post-partum. The cohort was 30% black, 65% white, 5% other; the mean patient age was 30 ± 6 years; and 88% were receiving beta-blockers and 81% angiotensin-converting enzyme inhibitors or angiotensin receptor blockers. An initial LVEF <0.30 ($p = 0.001$), an LVEDD ≥6.0 cm ($p < 0.001$), black race ($p = 0.001$), and presentation after 6 weeks post-partum ($p = 0.02$) were associated with a lower LVEF at 12 months. Survival free from major cardiovascular events (death, transplantation, or left ventricular [LV] assist device) was determined and 13% had major events or persistent severe cardiomyopathy.

Another study named “Pregnancy-related cardiovascular deaths in California: beyond peripartum cardiomyopathy” done by Hameed AB et al[16] concluded that obesity, african american race and substance abuse were important risk factors for pregnancy related cardiovascular deaths.this study retrospectively examined a case series of 64 cardiovascular pregnancy-related deaths from 2002 through 2006 and

found that 42 deaths caused by cardiomyopathy, and the pregnancy-related mortality rate from cardiomyopathy was 1.54 per 100,000 births.

In a study named “Maternal cardiovascular events during childbirth among women with congenital heart disease” conducted by A R Opotowsky et al[17] in 2011 in USA showed that Annual deliveries for women with CHD increased 34.9% from 1998 to 2007 compared with an increase of 21.3% in the general population. Women with CHD were more likely to sustain a cardiovascular event (4042/100,000 vs 278/100,000 deliveries). Arrhythmia, the most common cardiovascular event, was more frequent among women with CHD (2637/100,000 vs 210/100,000). Death occurred in 150/100,000 patients with CHD compared with 8.2/100,000 patients without CHD. Complex CHD was associated with greater odds of having an adverse cardiovascular event than simple CHD (8158/100,000 vs 3166/100,000). Pulmonary hypertension was associated with increased odds for an adverse cardiovascular event for CHD diagnoses of any complexity.

In 2010, Drenthen W et al[6], conducted a study in 1802 women with CHD, and 1302 completed pregnancies were observed. Independent predictors of cardiac, obstetric, and neonatal complications were calculated using logistic regression. The most prevalent cardiac complications during pregnancy were arrhythmias (4.7%) and heart failure (1.6%). Factors independently associated with maternal cardiac complications were the presence of cyanotic heart disease (corrected/uncorrected), the use of cardiac medication before pregnancy, and left heart obstruction. New characteristics were mechanical valve replacement (P = 0.0014), and systemic (P = 0.04) or pulmonary atrioventricular valve regurgitation related with the underlying (moderately) complex CHD (P = 0.03). A new risk score for cardiac complications is proposed. The most prevalent obstetric complications were hypertensive complications (12.2%). No correlation of maternal characteristics with adverse obstetric outcome was found.

The most prevalent neonatal complications were premature birth (12%), small for gestational age (14%), and mortality (4%). Cyanotic heart disease (corrected/uncorrected) (P = 0.0003), mechanical valve replacement (P = 0.03), maternal smoking (P = 0.007), multiple gestation (P = 0.0014), and the use of cardiac medication (P = 0.0009) correlated with adverse neonatal outcome.

In a study conducted by S S Brar et al[18] in 2007 in California named "Incidence, mortality, and racial differences in peripartum cardiomyopathy"showed that the overall incidence of PC was 1 in 4,025 deliveries. The incidence in whites, African-Americans, Hispanics, and Asians was 1 of 4,075, 1 of 1,421, 1 of 9,861, and 1 of 2,675 deliveries, respectively. The incidence of PC was greatest in African-Americans, which was 2.9-fold higher compared with whites (p = 0.03) and 7-fold that of Hispanics (p <0.001). With a mean follow-up of 4.7 years, the freedom from all-cause death was 96.7% by the Kaplan-Meier method. In conclusion, this large population-based study highlights important racial differences in the incidence of PC. We observed the lowest incidence of PC in Hispanics and the highest in African-Americans. Our finding also suggest that the current mortality associated with PC may be less than reported in older series, perhaps because of the high utilization of modern HF therapy.

In a study conducted by JAMES AH et al[19](2006) to determine the incidence, mortality, and risk factors for pregnancy-related acute myocardial infarction in the United States.A total of 859 discharges included a diagnosis of acute myocardial infarction, for a rate of 6.2 per 100,000 deliveries. Among these, there were 44 deaths, for a case fatality rate of 5.1%. Although acute myocardial infarction is a rare event in women of reproductive age, pregnancy increases the risk 3- to 4-fold.Single independent variables like age, race, and certain medical conditions and obstetric complications like Hypertension, thrombophilia, diabetes mellitus , smoking , transfusion , postpartum

infection and age 30 years and older remained as significant risk factors for pregnancy-related acute myocardial infarction.

In 2001 S C Siu et al[20] conducted a study on 562 consecutive pregnant women with heart disease and determined the outcomes of 599 pregnancies not ending in miscarriage. Pulmonary edema, arrhythmia, stroke, or cardiac death complicated 13% of pregnancies. Prior cardiac events or arrhythmia, poor functional class or cyanosis, left heart obstruction, and left ventricular systolic dysfunction independently predicted maternal cardiac complications; the cardiac event rate can be predicted using a risk index incorporating these predictors. Neonatal complications (20% of pregnancies) were associated with poor functional class or cyanosis, left heart obstruction, anticoagulation, smoking, and multiple gestations.

6.3 AIM AND OBJECTIVE OF THE STUDY

- To study the prevalence of heart diseases in pregnant women in north india.
- To study maternal cardiac and obstetric outcomes, fetal outcomes in pregnant women with heart diseases.
- To investigate the risk factors associated with morbidity and mortality in pregnant women with cardiovascular diseases.

7. MATERIALS AND METHODS

It will be a Single centre, Prospective observational study carried out on pregnant females with heart diseases from Cardiology and Obs & Gynae units in Dayanand Medical College and Hospital, a tertiary care hospital in Ludhiana, Punjab.

7.1 SOURCE OF DATA

The study population will consist of pregnant females meeting the inclusion criteria attending Cardiology & OBGY OPD and admitted patients in Cardiology & OBG wards/ICU's.

7.2 METHOD OF COLLECTION OF DATA

All women who will undergo cardiac assessment at baseline, when they first present for a cardiac assessment after presentation and followed there-after until 6 months postpartum will be included. Pregnant women who will undergo termination or have miscarriage (fetal death at <20 weeks gestation) will also be included. Baseline clinical, electrocardiographic, and echocardiographic variables collected at the time of the first antenatal visit and consultation with a cardiologist will include maternal age, parity, cardiac lesion, prior cardiac intervention, new york heart association (nyha) functional class, maternal cyanosis confirmed by oximetry, cardiac rhythm, ventricular systolic function, valvular function, and right ventricular systolic pressure. Systemic ventricular ejection fraction will be calculated using validated methods. Right ventricular systolic function will be visually evaluated as normal or impaired. Valvular area and gradients and severity of valvular regurgitation will also be measured using standardized criteria. All patients will be scored according to – CARPREG II risk scoring system

INCLUSION CRITERIA:

All pregnant women(>18 yrs of age and who are willing to participate in our study after signing informed consent) with following heart disease were included:-

1. Congenital HD
2. Rheumatic HD (Valvular)
3. LV Dysfunction
4. Cardiac arrhythmia(sustained/non sustained requiring treatment pre-pregnancy)
5. Cardiomyopathy
6. Thrombotic diseases
7. Prosthetic heart valves
8. Hypertensive disorders in pregnancy
9. Gestational Diabetes Mellitus
10. Any other acquired cardiac lesions.

EXCLUSION CRITERIA:

None.

DURATION OF STUDY:

18 months (Jan 2023 to June 2024)

OUTCOME

Cardiac outcomes

- Adverse maternal cardiac outcomes during antepartum, peripartum, and postpartum periods will be recorded up until the sixth postpartum month. All attempts will be made to ensure that patients make their visits prior to delivery and up to 6 months post-partum.
- **Primary cardiac outcomes** will be those utilized in the CAPREG II study - maternal cardiac death; cardiac arrest; sustained arrhythmia requiring treatment; left-sided heart failure (HF) defined as pulmonary edema; right-sided HF; stroke or transient ischemic

attack; cardiac thromboembolism; myocardial infarction; and vascular dissection.

- **Secondary cardiac outcomes** will be defined as a decline in NYHA functional class by ≥2 classes during the antepartum period. The need for urgent invasive treatment procedure/surgery during pregnancy and up to the 24th postpartum week, this cut off of 24 weeks postpartum will be set to exclude elective procedures that were deferred due to pregnancy.

Other outcomes

- The following data relating directly to the pregnancy will be collected: age at conception, cardiac complications, obstetric complications, perinatal complications, medication use, pregnancy duration, and mode of delivery.

- ***Obstetric complications*** will include intra-uterine growth retardation, pregnancy-induced hypertension [PIH, i.e. new-onset hypertension (>140/90 at two occasions) after >20 weeks of gestation], pre-eclampsia (PIH criteria plus >0.3 g proteinuria in the 24 h urine sample), eclampsia (pre-eclampsia with grand mal seizures), premature rupture of membranes (membrane rupture before onset of uterine contractions), premature labour (spontaneous onset of labour<37 weeks of gestation), post-partum haemorrhage (vaginal delivery >500 mL, caesarean delivery >1000 mL or requiring transfusion), and placental abruption and any other related complications.

- ***Perinatal complications*** will include foetal death (>22 weeks of gestation or >500 g), neonatal death (<30 days post-partum), still births ,and CHD in the baby of a mother with CHD.

STATISTICAL ANALYSIS:

Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate.All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

No.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Yes, Attached herewith.

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PROFORMA

S. No.

NAME

AGE

MRD NUMBER

CONTACT NUMBER

ETHINICITY

SOCIOECONOMIC STATUS

GESTATIONAL AGE AT ENROLLMENT

OBSTETRIC HISTORY

CHIEF COMPLAINTS

NYHA CLASS AT PRESENTATION

SMOKING OR TOBACCO USE

ALCOHOL USE

H/O IVF THERAPY

H/O PRIOR SURGERY/INTERVENTION

Past medical history

Mechanical valve prosthesis

Arrhythmia's

Pacemaker

Endocarditis

CHF

Stroke

Hypertension

Diabetes Mellitus

OTHER RISK FACTORS

PRESENT PREGNANCY COMPLICATIONS

Hypertension

GDM

Multiple pregnancy

Prothrombotic Events

Medication used prior to pregnancy

None

ACEI

Beta-blocker

Calcium-channel blocker

Digoxin

Anticoagulants

INJ Benzathine penicillin

EXAMINATION

BP HR SPO2 RR TEMP

ORODENTAL HYGEINE

PALLOR ICTERUS CYANOSIS CLUBBING EDEMA-FACIAL/PEDAL

CVS

CNS

CHEST

P/A

BODY MASS INDEX

INVESTIGATIONS

HB/PCV

TLC/DLC

PLATELET COUNT

PTI/PTTK

URINE ROUTINE

UREA/CREATININE

BILIRUBIN(T/D)

SGOT/SGPT

TOTAL PROTEIN/ SERUM ALBUMIN

SERUM LDH

BNP LEVELS

RBS

BLOOD GROUPING

USG WHOLE ABDOMEN AND FETAL WELL BEING

FETAL ECHO IF DONE

OTHERS

Maternal congenital lesion

Aortic valve stenosis/bicuspid aortic valve

Surgically repaired coarctation

TOF repaired

Pulmonary valve stenosis

VSD

ASD

AV septal defect

Abnormal pulmonary venous return

Marfan syndrome

Other aortopathy

D-TGA post atrial switch

D-TGA post arterial switch

CCTGA

Fontan physiology

Other complex CHD

Unrepaired lesions

Eisenmenger physiology

VALVULAR DISEASE

MITRAL

TRICUSPID

AORTIC

PULMONARY

Echo cardiograph parameters

Mitral valve regurgitation

Mitral valve stenosis

Tricuspid valve regurgitation

Tricuspid valve stenosis

Pulmonary valve stenosis

Pulmonary valve regurgitation

Aortic valve stenosis

Aortic valve regurgitation

Systemic ventricular dysfunction

Right ventricular dysfunction(TAPSE)

Pulmonary hypertension(PASP)

REGIONAL WALL MOTION ABNORMALITIES

ECG FINDINGS

ST ELEVATION

ARRYTHMIAS

Coronary Angiography DONE (if any)

Any other cardiac intervention (if any)

-BMV

Maternal mortality-yes/no

Maternal morbidity

Heart failure

Supraventricular arrhythmias

Ventricular arrhythmias

Cardiac thromboembolism

Obstetric complications

PIH (Eclampsia or pre-eclampsia)

Post-partum haemorrhage

Mode of delivery (C SECTION, VAGINAL, INSTRUMENTAL)

Antenatal corticosteroids

Others

Fetal complications

APGAR score

Gestational age of delivery

NICU admission

Fetal death

Neonatal death

Mean birth weight

Fetal CHD

Others

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I……..D/o, W/o, S/o…… ……am willing to participate in the study **"PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

……. ……

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

“PREVALENCE, CLINICAL PROFILE, RISK FACTORS & MATERNO-FETAL OUTCOMES IN PREGNANT FEMALES WITH CARDIOVASCULAR AILMENTS IN A TERTIARY CARE CENTRE IN NORTH INDIA” ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ